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Drinking Water Criteria Document for Radium (Draft)

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CRITERIA DOCUMENT FOR RADIUM IN DRINKING WATER

April 1985

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CRITERIA AND STANDARDS DIVISION (WH-550)
OFFICE OF DRINKING WATER
U.S. ENVIRONMENTAL PROTECTION AGENCY
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The Office of Drinking Water (ODW), U.S. Environmental Protection Agency has prepared a Drinking Water Criteria Document on Radium This Criteria Document is an extensive review of the following topics:

- Physical and chemical properties of Radium
- Toxicokinetics and human exposure to Radium
- Health Effects of Radium in humans and animals
- Mechanisms of toxicity of Radium
- Quantification of toxicological effects of Radium

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I. SUMMARY

Two isotopes of radium, found in the naturally occurring uranium and thorium series, are of importance in relation to public health. These are ^{226}Ra, a 1,620-year half-life isotope which is a progeny of ^{238}U, and ^{228}Ra, a 5.75-year half-life isotope which is a progeny of ^{232}Th.

The geochemistry of the different elements in the naturally occurring uranium and thorium series is element specific and is related to the geological concentration of the parent and the related half-lives. Radium-228 concentrations in water depend on the geochemistry of the highly insoluble parent ^{232}Th. Radium-226 concentrations are related to the properties of the several parents. Thorium distributions in the geosphere are controlled by primary geochemical processes (such as magnetic crystallization) or secondary physical processes (such as sedimentary enrichment of thorium-bearing methods in placer deposits). Uranium has two oxidation states, and the +6 state (uranyl) can form highly soluble complexes which can be transported long distances by oxidizing groundwater before being removed by adsorption or reduction to the +4 state.

Analytical methods have been developed and verified for the determination of concentration of ^{226}Ra and ^{228}Ra in water, as well as for gross alpha particle activity. Methods for radium measurements include the radon-emanation technique, barium sulfate precipitation, direct alpha particle measurements, alpha-gamma and beta-gamma coincidence techniques and direct gamma ray measurement.

Radium-226 and -228 are both bone volume seekers. They can deposit via a surface exchange mechanism or a self burial mechanism. If radium is ingested continuously, the amount in bone will be 25.0 times that ingested daily for ^{226}Ra and 9.2 times that ingested daily for ^{228}Ra. The f_1 value is approximately 0.2 (fraction of that ingested that goes to blood).

Radium is found primarily in groundwater supplies (no non-compliance with the interim regulation has been reported for a surface water supply). From the available data, there are two specific geological regions where over 75% of the known radium violations of the interim regulations occurred:

a. The Piedmont and Coastal Plain provinces in New Jersey, North Carolina, South Carolina and Georgia.

b. A north central region consisting of parts of Minnesota, Iowa, Illinois, Missouri and Wisconsin.

It has been estimated that approximately 500 systems will be found to exceed the maximum contaminant level (MCL) of 5 pCi/L after the initial monitoring is complete.

The existing data for occurrence of ^{228}Ra in drinking water supplies are not nearly as well known as those for ^{226}Ra. A geochemical model developed for the occurrence of ^{228}Ra predicts that three main aquifer types are likely to produce groundwater with relatively elevated ^{228}Ra activities: granite rock aquifer, arkosic sand and sandstone aquifers and quartzose sandstone aquifers.

Health effects studies for ingested radium have been performed with rodents and beagle dogs. They all indicate deaths that result from bone sarcomas.

Human health effects due to ingested radium have been found among the radium watch dial painters. The most important are bone sarcomas and head carcinomas. Using the dose-response data from such human studies, it has been estimated that lifetime ingestion of water containing 5 pCi of ^{226}Ra per liter can produce somewhere in the range of $2\text{-}4 \times 10^{-5}$ sarcomas per lifetime per person. The shape of the doseresponse curve is not known for lower levels of concentration such as 5 pCi/L, but at higher levels, the curve could be linear (a quadratic shape cannot be ruled out). Risk rates have been estimated for both bone and head sarcomas.

The effective dose equivalent rate and risk rate due to ingestion of ^{226}Ra and ^{228}Ra have been estimated using a modified International Commission on Radiological Protection

(ICRP, 1979) dosimetric model and organ weighting factors derived from the BEIR III (NAS 1980) report. The results of these calculations are shown in Table I-1.

Table I-1 Lifetime Risk Rates and Annual Effective Dose Equivalent Rates as They Relate to Concentration of Radium in Drinking Water[a]

Lifetime individual risk rate	Annual effective dose equivalent rate (mrem/year)	Drinking water concentrations[b] (pCi/L)	
		^{226}Ra	^{228}Ra
10^{-3}	100	100	200
10^{-4}	10	10	20
10^{-5}	1	1	2
10^{-6}	0.1	0.1	0.2

[a] Assuming an ingestion rate of 2 L/day.

[b] Rounded off to one significant figure and involving an uncertainty of a factor of four to five.

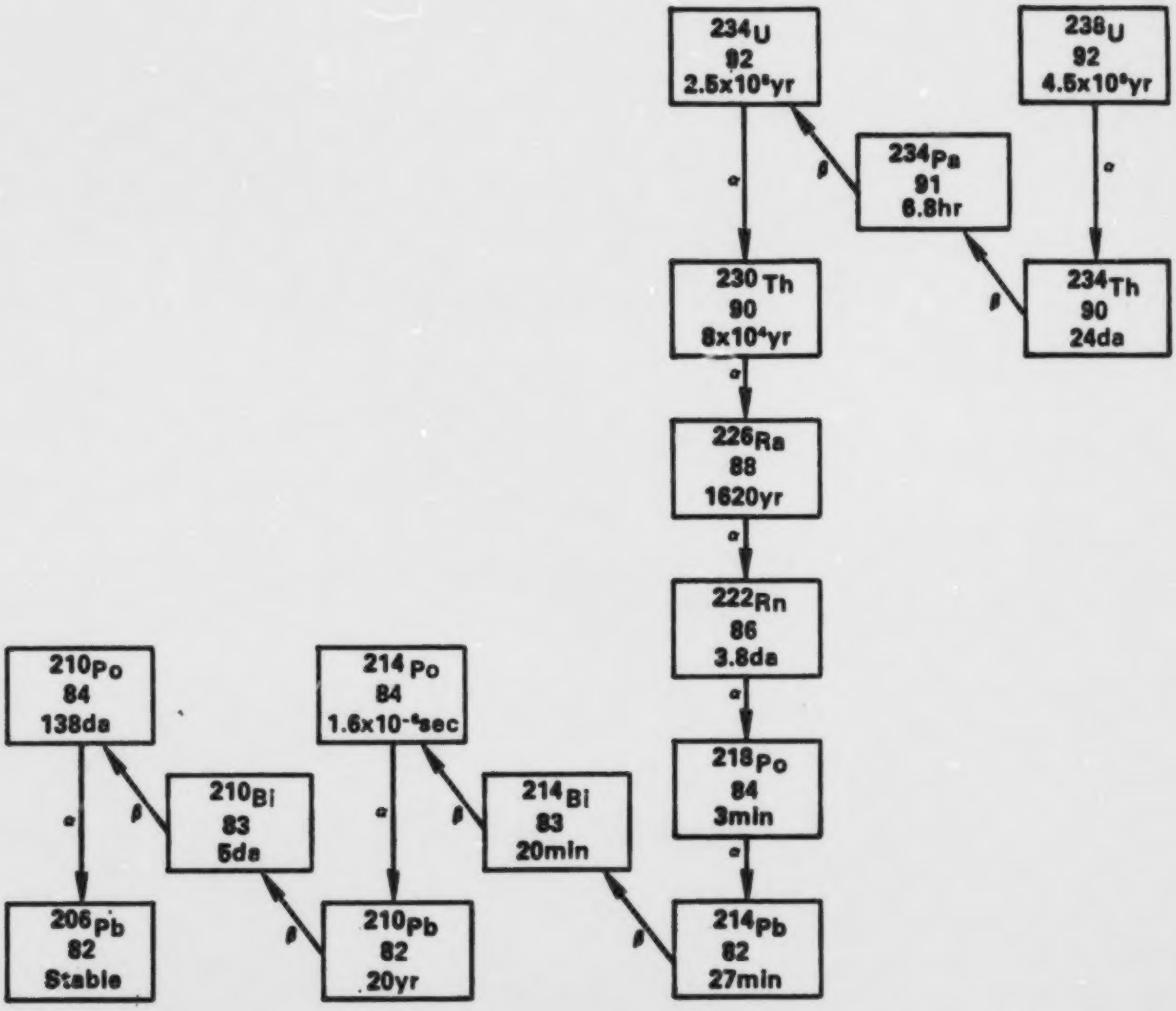
II. PHYSICAL CHARACTERISTICS, CHEMICAL PROPERTIES AND ANALYTICAL METHODS

Radium is an alkaline earth element which has 13 known radioisotopes, although only four are natural, and only three of these four are of potential environmental concern. Radium-226 is a member of the uranium decay series and has a half-life of 1,620 years (Figure II-1). Radium-226 decays by emission of an alpha particle to form the gaseous element ^{222}Rn, a process with important consequences, both analytically and in terms of its human health impacts. Radium-226 is the isotope which is most commonly referred to as radium and for which there exists the most data on natural occurrence and health effects. Radium-228 and ^{224}Ra are both members of the thorium decay series (Figure II-2). Radium-228 is the first progeny of ^{232}Th and is a weak beta emitter with a 5.7-year half-life. Radium-224 is an alpha emitter which has a half-life of only 3.6 days. Because all three isotopes are progeny of long-lived, natural radionuclides, they have special chemical and radiological properties that are important in understanding their behavior, especially as it relates to occurrence in drinking water.

A. Radiological Decay Processes

All of the natural radioactive decay series begin with a long-lived parent radionuclide. These parent nuclides undergo

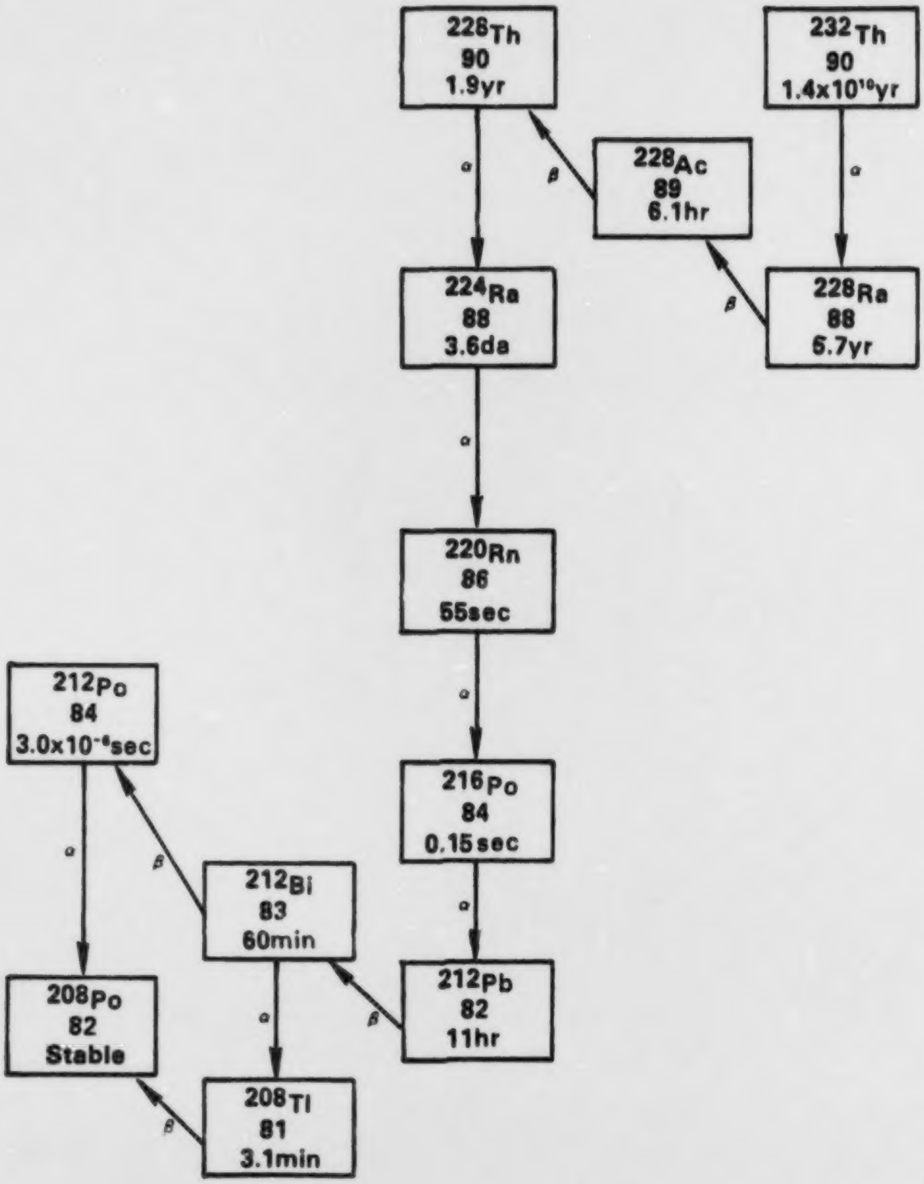
THE URANIUM SERIES



^{234}U 92 2.5×10^5yr
^{238}U 92 4.5×10^9yr
^{234}Pa 91 6.8hr
^{230}Th 90 8×10^4yr
^{234}Th 90 24da
^{226}Ra 88 1620yr
^{222}Rn 86 3.8da
^{210}Po 84 138da
^{214}Po 84 1.6×10^{-4}sec
^{218}Po 84 3min
^{210}Bi 83 5da
^{214}Bi 83 20min
^{206}Pb 82 Stable
^{210}Pb 82 20yr
^{214}Pb 82 27min
α
β

Figure II-1

THE THORIUM SERIES



228Th 90 1.9yr
232Th 90 1.4x10¹⁰yr
α
β
228Ac 89 6.1hr
α
224Ra 88 3.6da
β
228Ra 88 5.7yr
α
220Rn 86 55sec
212Po 84 3.0x10⁻⁶sec
α
216Po 84 0.15sec
α
β
212Bi 83 60min
α
208Po 82 Stable
α
β
212Pb 82 11hr
208Tl 81 3.1min
β

Figure II-2

spontaneous transformations that result in the emission of particles plus radiant energy and the formation of a radioisotope of a different element. This process is repeated until a stable isotope of lead is formed. If a parent nuclide is removed from its progeny and deposited elsewhere, it will produce the complete series of nuclides in a length of time determined by the half-life ($t_{1/2}$) of the longest lived progeny. If there is no loss or gain of the progeny nuclides by geologic processes during this time, the series is described as being in secular equilibrium. Equilibrium is attained when the progeny product decays at the same rate as it is produced from the parent nuclide. The progeny product grows and reaches equilibrium in a period of time equal to about five half-lives of the progeny. For example, in a closed system, the $^{234}U/^{238}U$ activity ratio will be 1.0 (at equilibrium) after approximately one million years, whereas the $^{210}Po/^{226}Ra$ activity ratio will be 1.0 after nearly one hundred years. If the progeny isotope is physically separated from its parent, it will also decay to 3% of the initial value in five half-lives. Thus, ^{228}Ra, when removed from ^{232}Th by dissolution into water, will decay to half its original activity in 5.7 years and to 3% of its initial activity in about 30 years. In contrast, ^{226}Ra, when removed from its parent, will decay to 1.4% in about 10,000 years.

Secular equilibrium is attained only when there is no loss or addition of a nuclide in the decay series. However, in groundwater, it is common to find disequilibrium between parent-progeny pairs, and the degree of disequilibrium can be very large. There are several mechanisms by which disequilibrium or isotopic fractionation occurs:

1. Separation of nuclides can occur when the progeny differs chemically from the parent. An extreme example is the generation of ^{222}Rn, an inert gas, from ^{226}Ra, a solid. The greater mobility of the radon isotope in water may result in very large values for the ^{222}Rn/^{226}Ra activity ratio which can approach 10^6. Another important example is the extreme insolubility of thorium relative to uranium in an oxidizing environment. Therefore, ^{230}Th generated by the decay of ^{234}U in groundwater is rapidly removed by sorption onto the aquifer solids.

2. Progeny isotopes of the same element are more vulnerable than parents to dissolution due to the "Szilard-Chalmers" effect of alpha recoil (Cherdyntsev 1971). Alpha particles emitted during decay release enough energy to cause radiation damage to the crystal lattice and/or displacement of the daughter atoms from their original position in the mineral. The progeny atom becomes embedded in a foreign part of the mineral, where it is more weakly bound than the parent. The progeny then migrates into dislocation zones, where

it is more susceptible to dissolution. For example, ^{230}Th, as a progeny of ^{234}U, is much more soluble than ^{232}Th. Alpha recoil can also cause the direct transfer of the progeny into an aqueous phase. Furthermore, in the case of uranium isotopes, ^{234}U is more likely to be oxidized to the +6 state, which is more mobile than U^{4+}.

These mechanisms are important in understanding the radiochemical behavior of radium isotopes, which occur only by decay of parent radionuclides. Radium-226 is a fifth generation progeny with three events of alpha decay (and recoil). Although ^{226}Ra is a progeny of uranium, its immediate parent is ^{230}Th, a highly insoluble element. Therefore, dissolution of ^{226}Ra occurs only by desorption or alpha recoil. These processes are enhanced by the fact that in an aquifer, ^{230}Th produced by decay of ^{234}U in solution tends to adsorb onto the surface of the aquifer solids, a location from which ingrown ^{226}Ra is more likely to dissolve. Actually, radium in water would not necessarily be related to the uranium content of the water or the aquifer solids. With the relatively long ^{230}Th half-life (75,000 years), ^{230}Th could accumulate onto the solids while the uranium passes through, resulting in higher ^{226}Ra than uranium in solution. In comparison, because ^{228}Ra is the immediate progeny of ^{232}Th by alpha decay, its distribution will be directly related to the distribution of ^{232}Th in the aquifer solids.

B. Geochemistry of Radium Isotopes

The distribution of ^{228}Ra and ^{226}Ra in water is a function of the thorium and uranium content of the aquifer, the geochemical setting of the aquifer solids and the half-life of each isotope. Specific geological and chemical processes that control the thorium and uranium content in aquifers are discussed in detail by Olson and Overstreet (1964), Cherdynstev (1971) and Gableman (1977). In fact, thorium and uranium behave similarly, with one important exception that is most responsible for their eventual separation. Thorium has one oxidation state and is immobile at low temperatures. Therefore, thorium distribution is controlled by primary geochemical processes (such as magnetic crystallization) or secondary physical processes (such as sedimentary enrichment of thorium-bearing minerals in placer deposits). Uranium has two oxidation states, and the +6 state (uranyl) can form highly soluble complexes which can be transported long distances by oxidizing groundwater before being removed by adsorption or reduction to the +4 state. The estimated average crustal Th/U activity ratio is 1.2:1.5, so that, in the absence of enrichment or depletion processes, ^{228}Ra activity should be higher than that of ^{226}Ra. However, the tendency for uranium enrichment under certain geochemical conditions results in regions of higher ^{226}Ra, which accounts for EPA's decision to emphasize ^{226}Ra in the interim regulations.

Radium enters groundwater by dissolution of aquifer solids, by direct recoil across the liquid-solid boundary during its formation by radioactive decay of its particular thorium parent in the solid and by desorption. As discussed earlier, the mechanism of alpha recoil is an important factor in the higher solubility of progeny isotopes compared with their parent isotopes. Uranium-234/uranium-238 activity ratios in groundwater are generally greater than 1.0 and can be as high as 28 (Gilkeson and Coward 1982). Radium-224/radium-228 activity ratios in South Carolina groundwater range from 1.2 to 2.0 (Hess et al. 1983), and in Connecticut from 0.8 to 1.7 (Kriege and Hahne 1982).

All isotopes of radium have the same chemical behavior, which is only slightly affected by their differences in half-life. Radium has chemical properties similar to the other alkaline earth elements, particularly barium and calcium. It has only one oxidation state (+2) in solution. Because of its highly basic character, the divalent ion is not easily complexed (Kirby and Salutsky 1964). Although some radium compounds are very insoluble in water, their solubility limits are orders of magnitude above the concentrations of radium that occur naturally in water, which are usually below 10^{-12} g/L. Rather, radium's solubility appears to be controlled by two factors: (1) dissolution, which is controlled by the distribution of its parent nuclide, especially in the aquifer solids for groundwater and (2) removal by sorption processes. There will be large differences in the solubility of a radium atom formed from decay of a uranium or thorium parent which is tightly bound in a crystalline lattice, such as in a monazite in granite, versus that adsorbed onto the surface of a sand grain. During decay, the recoiling radium atom is more likely to enter solution from the surface of a sand grain or clay particle than to migrate out of a rigid crystal structure through small fractures in the rock to the groundwater. Therefore, the initial solubilities of ^{228}Ra and ^{226}Ra are very much a function of how their parent uranium and thorium are distributed. But radium can be rapidly removed from solution, primarily by nonspecific adsorption and coprecipitation. For example, dissolved radium concentrations can be reduced significantly in waters which contain iron hydroxide, an efficient scavenger of radium. Another efficient scavenger of radium is barium sulfate, which is frequently used in purification of radium in the laboratory. Studies of groundwaters in Illinois which exhibit high barium concentrations have shown that radium concentrations are affected by dissolution/ precipitation of secondary barite, particularly when mixing of groundwater from different aquifers occurs due to variations in the rate of pumping (Gilkeson and Coward 1983).

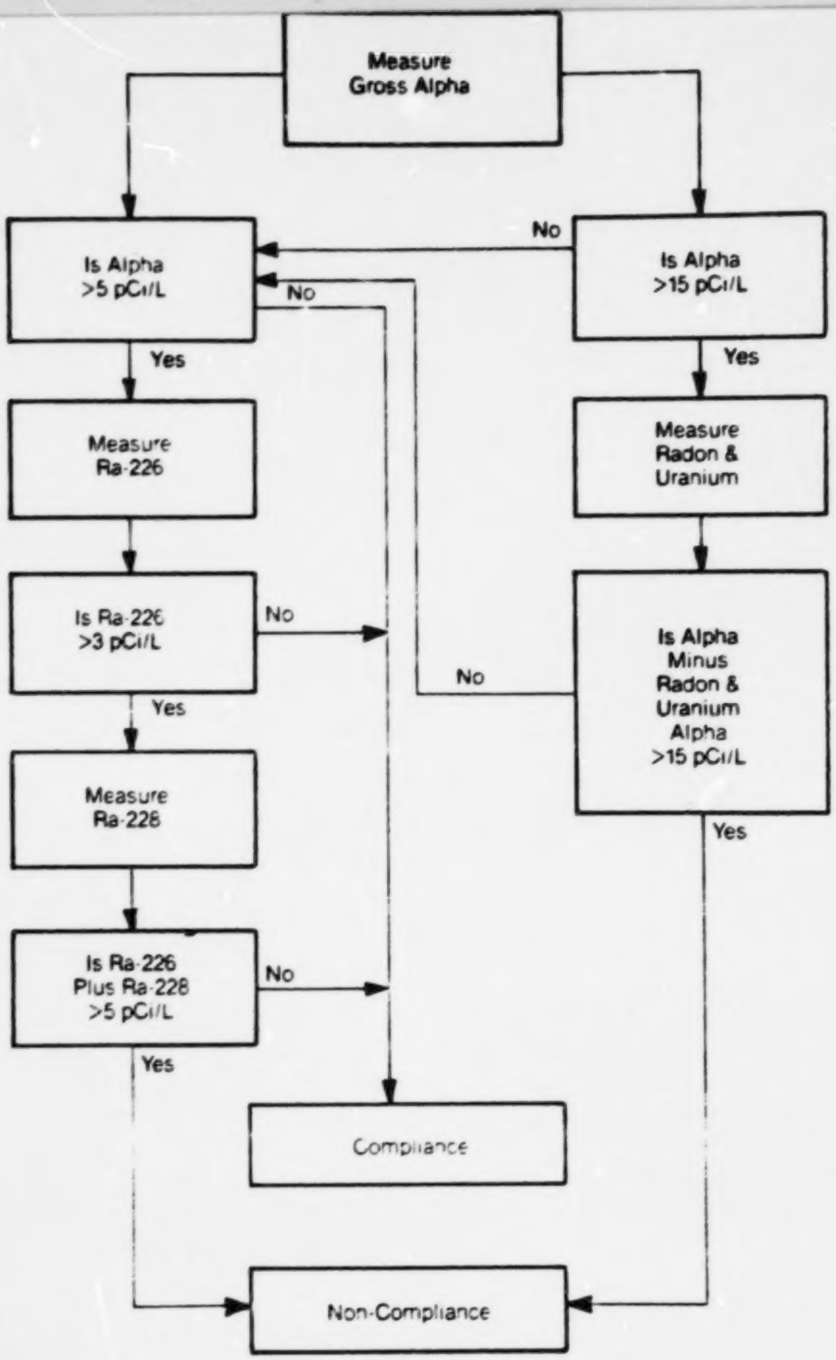
Recent studies have provided some information on the rates of radium removal from groundwater. King et al. (1982) proposed that the distance of radium transport in groundwater was less than that of 222Rn ($t_{1/2}$ = 3.8 days) due to continual adsorption of radium onto the aquifer solids. Krishnaswami et al. (1982) calculated adsorption and desorption rate constants for radium in Connecticut aquifers and proposed that radium removal rates are rapid (as short as a few minutes). Equilibrium between adsorption and desorption is also quickly established, but Krishnaswami et al. (1982) concluded that the partition coefficient strongly favored the solid phase, and almost all radium introduced into the groundwater studied resided on particle surfaces in the adsorbed state. However, the extent of sorption is controlled by the geochemical reactivity of the aquifer material. King et al. (1982) noted that the average ^{228}Ra and ^{226}Ra activity in the crystalline aquifers of South Carolina was lower than that in the Coastal Plain sediments, even though the thorium and uranium content of the rock aquifers was higher. Furthermore, the ^{222}Rn activity in the crystalline aquifers was ten times greater than that in aquifers sampled in the Coastal Plain. King et al. (1982) concluded that the affinity of radium for adsorption sites in the fresh rock surfaces, which have higher cation-exchange capacities, was greater than its affinity for the sand and gravel deposits composed of refractory minerals such as quartz. Thus, radium in groundwater does not accumulate with groundwater transport in aquifers. Instead, it stays very close to the area in which it is produced, except when the groundwater has a dissolved solids concentration greater than 1,000 mg/L.

The insolubility of radium and thorium can be inferred from studies of potential contamination of groundwater due to seepage from uranium tailings ponds in New Mexico (Kaufmann et al. 1976). At one such pond, it was estimated that nearly 3 x 10^9 L of seepage entered the shallow aquifer over a 20-year period. The wastes in this pond contained approximately 200 pCi/L of ^{226}Ra and 166,000 pCi/L of ^{230}Th. Thus, nearly a Ci of ^{226}Ra and 500 Ci of ^{230}Th were available to leach into the shallow groundwater. Yet, in 1975, monitoring wells located one kilometer downgradient from the pond showed no evidence of contamination.

C. Analytical Methods

1. Introduction

The interim regulations specified a sampling and analytical protocol which was aimed at reducing the burden of monitoring for radionuclides. Figure II-3 shows the analytical flowchart. All community water supplies were to be sampled for measurement of the gross alpha particle activity. The results were to be reported from the single analysis of an annual composite of four consecutive quarterly samples or from an average of analyses of four consecutive quarterly samples. The gross alpha particle activity measurement served two functions: (a) as a screen, with measurement of ^{226}Ra required only if the gross alpha particle activity exceeded 5 pCi/L, and


Measure Gross Alpha
Is Alpha >5 pCi/L
Is Alpha >15 pCi/L
No
No
Yes
Yes
Measure Ra-226
Measure Radon & Uranium
Is Ra-226 >3 pCi/L
No
Is Alpha Minus Radon & Uranium Alpha >15 pCi/L
No
Yes
Measure Ra-228
Yes
Is Ra-226 Plus Ra-228 >5 pCi/L
No
Yes
Compliance
Non-Compliance

---

[a] It is not a requirement that radon and uranium be measured if the gross alpha particle activity is less than 15 pCi/L.

Adapted from USEPA, Las Vegas, NV, Environmental Monitoring and Support Laboratory.

Figure II-3 Flow Chart for Gross Alpha Particle Activity Monitoring[a]

(b) for determining compliance, because the MCL was set at 15 pCi/L.

There was a second screening step in the analytical protocol specified in the interim regulations. Only when ^{226}Ra exceeded 3 pCi/L was the measurement of ^{228}Ra required. This screen was proposed for two reasons. Because the approved methods for ^{228}Ra are time-intensive and costly, requiring ^{228}Ra analysis for every sample was considered unduly expensive. Furthermore, it was believed that the largest contributor to radium violations of the MCL would be ^{226}Ra. The regulations encouraged states to develop additional analytical requirements where ^{228}Ra was a potential problem. Follow-up monitoring of all systems was required every four years.

2. Analytical Methods for Gross Alpha Particle Activity

The measurement of the gross alpha particle activity is the most important of all the analytical requirements because it is conducted on all samples and used as a screen for additional analyses. The method is simple: an aliquot of a preserved drinking water sample is evaporated to dryness, weighed and counted with an alpha-scintillation detector system. Both the laboratory technique and the counting equipment required for this method are readily found in water quality laboratories.

In spite of the ease of the technique, there are still several serious problems. Because of the relatively large size and very low energy of alpha particles, self-absorption significantly reduces the sensitivity of the measurement for samples with high dissolved solids. The prescribed procedures state that the density of the sample residue should not exceed 5 mg/cm^2 (Krieger and Whittaker 1980). Thus, for a sample containing 500 mg/L dissolved solids, with an instrument counting efficiency of 8% and an alpha background of 1.0 counts per minute (cpm), the suggested aliquot of 100 mL would need to be counted for 700 minutes to meet the required sensitivity of 3 pCi/l. The use of an anticoincidence system can reduce background to 0.1 counts/min and thus reduce the counting time. Many samples have at least 500 mg/L dissolved solids, and such long counting times seem unreasonable for routine sample analyses. In practice, instead of using long counting times, the results are often reported with very large uncertainties.

The National Interim Primary Drinking Water Regulations (NIPDWR) (USEPA 1976a) addresses these problems of sensitivity and uncertainty only briefly, indicating in Section 141.25(c) that the listed detection limit is the concentration which can be counted with a precision of ±100% at the 95% confidence limit (1.96 times the standard deviation of the net counting rate of the sample). Additional information on sensitivity, accuracy and uncertainty is given in the EPA's prescribed procedures (Krieger and Whittaker 1980) and is based on collaborative analyses of factors conducted on three samples by 18 laboratories. A more realistic and empirical determination of the detection limit could be made by analysis of the results from EPA's laboratory crosscheck program.

To determine compliance with the MCL for the gross alpha particle activity, radon and uranium activities are excluded. The uranium is measured fluorometrically or radiochemically, and ^{238}U is assumed to be in radioactive equilibrium with ^{234}U. For many systems with high gross alpha particle activities (greater than 25 pCi/L), it is frequently not possible to account for all the activity by separate analysis of ^{226}Ra and uranium. There is always great concern about what constitutes the remaining alpha activity although some is ^{226}Ra that has been trapped in the evaporated solids. Some of these samples have been analyzed by alpha spectroscopy to measure the activity of specific alpha emitters (such as ^{232}Th, ^{230}Th, ^{210}Po and the actual ^{234}U/^{238}U), and in most cases, the sum of the individual activities does not equal the gross alpha particle activity.

Improvements in the gross alpha particle activity method have been suggested (Blanchard et al. 1983). For samples with high dissolved solids, EPA is currently evaluating a barium sulfate/iron hydroxide coprecipitation technique (Lieberman 1982) which alleviates self-absorption interferences. The uncertainty in the method could be reduced even for samples with low dissolved solids by determination of self-absorption factors for the more common alpha particle activity detectors. To assess the reliability of the results, detection limits and the uncertainty of methods applied under normal conditions should be reported by each laboratory.

3. Radium-226 Analytical Methods

The approved method for analysis of ^{226}Ra in water samples is called the "radon-emanation technique" (Krieger and Whittaker 1980). The ^{226}Ra is concentrated and separated by coprecipitation with barium sulfate. The precipitate is dissolved in EDTA reagent, which is sealed in a bubble flask and stored for ingrowth of ^{222}Rn for at least four days. The ingrown radon gas is purged from the solution and transferred to a scintillation cell. After about four hours, when the ^{222}Rn progenies have reached equilibrium, the scintillation cell is counted for alpha activity. Note that in this technique, the water sample is not consumed. Only the ingrown ^{222}Rn is milked from the sample, and it can be repeatedly milked for additional analyses without destruction of the sample. Also, there are no radioactive interferences in this method. It has a very low detection limit, as low as 0.1 pCi/L. The main analytical problems come from maintaining current background for the scintillation cells and from variations in purging efficiency among samples.

Statistical evaluation of five intercomparison studies for ^{226}Ra in water using the radon-emanation technique showed that intralaboratory error ranged from 14% to 19%, with an average of 10.2% (Krieger and Whittaker 1980). The coefficient of variation for systematic error among the total of 174 laboratories ranged from 14% to 18%, with an average of 16.2% for the five studies. Most importantly, the coefficient of variation for the total error among laboratories based on a single analysis ranged from 16% to 20%, with an average of 19.4% for the five studies.

Although there are no serious analytical problems with the radon-emanation technique, other methods have been developed and proposed (Noyce 1981). The EPA has prescribed a technique for determination of all alpha-emitting radium isotopes. It follows the above technique, but uses a second barium sulfate precipitation and alpha counting of the solid (Krieger and Whittaker 1980). Some alternative techniques simultaneously or sequentially measure ^{228}Ra, a very desirable capability. These proposed methods are discussed in the section on ^{228}Ra determinations because most were developed as alternatives to the approved methods for ^{228}Ra.

4. Radium-228 Analytical Methods

Historically, the accurate measurement of ^{228}Ra in environmental samples has been difficult. Radium-228 itself decays by emission of a weak beta particle, which is difficult to detect. The EPA-approved method utilizes the stronger beta decay of ^{228}Ac, the immediate progeny of ^{228}Ra, in a manner similar to measuring ^{222}Rn ingrown from ^{226}Ra. The shorter half-life of ^{228}Ac requires storage of the purified Ba(Ra)SO_4 for only 36 hours. The ingrown ^{228}Ac is then carried on yttrium oxalate, purified and beta-counted on a gas-flow proportional counting system (Krieger and Whittaker 1980). Besides being time-intensive and costly, this technique is inaccurate and imprecise, as is shown by results from EPA intercomparison and crosscheck programs. Tables II-1 and II-2 show the high frequency of unacceptable ^{228}Ra results from intercomparison and performance tests conducted under normal operating conditions by nationwide laboratories (Blanchard et al. 1983).

Another problem with the ^{228}Ra method is the possibility for contamination by the ^{90}Y daughter of ^{90}Sr. Tables II-1 and II-2 show the interference when the samples are analyzed by the approved method. Performance samples in Table II-1 contain a complex mixture of radionuclides, including ^{90}Sr-^{90}Y. Intercomparison samples in Table II-2 contain only ^{226}Ra and ^{228}Ra plus their progeny, except when 26 pCi of ^{90}Sr/L was added in the 9/79 study to examine the effects of ^{90}Y daughter interference.

Other methods that are becoming available appear to be

Table II-1 Summary of Ra-228 in Water Analyses in the EMSL-LV Performance (Blind) Sample Studies

Study	Ra-228 known value (pCi/L)	Ra-228 grand average (pCi/L)	Sr-90 (pCi/L)	No. of laboratories		% Laboratories acceptable
				Participated	Acceptable	
4/78	7.9	10.9 ± 4.8	10.0	23	6	26.1
10/78	5.4	6.6 ± 2.1	5.0	25	14	56.0
4/79	6.2	8.1 ± 4.2	8.0	31	11	35.5
10/79	0.0	4 ± 2	7.0	21	--	--
4/80	21.3	18.7 ± 3.2	0.0	32	22	68.8
10/80	9.2	8.5 ± 2.1	0.0	33	23	68.7
4/81	12.0	16.4 ± 8.5	28.0	41	18	43.9
10/81	9.2	10.3 ± 3.1	14.4	42	22	52.4
		Samples containing Sr-90, average				42.8 ± 12.2
		Samples not containing Sr-90, average				68.7 ± 0.7

Table II-2 Summary of Ra-228 in Water Analyses in The EMSL-LV Intercomparison (Cross-Check) Sample Studies

Study	Ra-228 known value (pCi/L)	Ra-228 grand average (pCi/L)	No. of laboratories Participated	No. of laboratories Acceptable	% Laboratories acceptable
9/78	20.8	18.0 ± 6.0	20	12	60.0
12/78	8.9	9.6 ± 2.8	23	10	43.5
3/79	13.6	13.1 ± 4.2	24	15	62.5
6/79	14.4	13.1 ± 4.6	28	13	46.4
9/79[a]	12.7	16.4 ± 5.9	33	10	30.3[b]
12/79	1.0	1.8 ± 1.3	18	14	77.8
3/80	2.6	3.5 ± 1.9	34	13	38.2
6/80	1.7	2.4 ± 1.3	29	13	44.8
9/80	14.4	12.9 ± 3.6	33	17	51.5
12/80	10.5	10.3 ± 3.7	37	24	64.9
3/81	7.3	7.0 ± 1.6	46	34	73.9
6/81	8.0	7.7 ± 2.8	51	30	58.8
9/81	11.7	10.9 ± 3.1	49	33	67.3
12/81	9.0	9.1 ± 1.7	46	39	84.8
3/81	10.2	10.3 ± 3.0	55	39	70.9
6/82	8.7	8.7 ± 1.9	53	39	73.6
9/82	11.0	10.6 ± 2.3	55	42	76.4
				Average	62.2 ± 14.0[c,d]

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[a] In this study, 25 pCi of Sr-90/L was added to demonstrate the Sr-90 influence on the methods being used.

[b] This value was not used in the average calculation.

[c] Average of % acceptable labs for first eight studies (2 years) is 53.1 ± 13.0%.

[d] Average of % acceptable labs for second eight studies (2 years) is 71.3 ± 7.9%.

superior to the approved methods (Noyce 1981) and should be evaluated. Many of them simultaneously determine ^{226}Ra. One such method uses beta-gamma coincidence counting after radium coprecipitation with barium sulfate (McCurdy and Mellor 1981). This method requires a 1-L sample and a minimum of chemical techniques in order to meet the required sensitivity of measurement for ^{226}Ra. The instrument consists of a well-type NaI(Ti) detector for gamma rays, a thin plastic scintillator for detecting beta particles and a coincidence pulse analyzer.

Another method's procedures consist of coprecipitating radium with barium sulfate, then dissolving the precipitate in alkaline EDTA, mixing the solution with liquid scintillator solution and counting the low-energy beta particles of ^{226}Ra (Asikainen 1981). The author claims adequate sensitivity with 1-L samples. A modification of this method has been tested by EPA Environmental Monitoring Systems Laboratory in Cincinnati (EMSL-CIN).

A third method specifies a 3-L sample and the same radium barium coprecipitation procedure described above. The precipitate is collected on a filter, allowed two days for ^{228}Ac ingrowth and then analyzed for ^{228}Ra with a gamma ray spectrometer. The 986-keV gamma ray emitted by the ingrown ^{228}Ac is measured. The state of Arkansas is seeking approval for use of this method (Henry 1982).

In a fourth method, radium is collected from a multi-liter sample on a column of fibers coated with manganese dioxide (Michel et al. 1981). Radium is then dissolved from the column, coprecipitated with barium sulfate, collected on a filter paper and analyzed for ^{228}Ra by counting gamma rays emitted by the ingrown ^{228}Ac, as in the above-cited method. After a period of weeks to months, the ingrown ^{228}Th plus ^{224}Ra can also be measured with an alpha particle detector. As an alternative, the fibers are compressed, and ^{228}Ac is counted directly with a gamma ray spectrometer.

The interim regulations required repeated monitoring at four-year intervals. At the National Workshop on Radioactivity in Drinking Water, it was recommended that data be examined to determine whether this interval was appropriate (Blanchard et al. 1983). However, there have been several studies on the temporal variability of the activity of radium isotopes in groundwater systems. Kriege and Hahne (1982) reported that the mean value for the average percent deviations for 141 samples collected over a period of 18 years in Iowa was 21% with a relative standard deviation of 15%. Michel et al. (1981) found a maximum variation of 19% over two years in individual wells. Therefore, in single-well systems, one sample should be representative of the average annual activity. The present requirement for monitoring at four-year intervals would not be necessary unless changes to the system have been made. Systems with multiple wells have the potential for continuously variable radium concentrations due to the relative contribution of each well when sampled.

5. Summary

There are several issues that need to be addressed before improvements in analytical techniques for measuring radium isotopes in drinking water can be realized. The most important of these issues concerns the rationale for the use and levels of screening procedures. To date, only two studies have evaluated the efficiency of the gross alpha particle activity screen in detecting ^{226}Ra violations. The author(s) of one study, in which a large number of samples from Iowa were analyzed, concluded that a screening level of 2 pCi/L for gross alpha particle activity was required for samples with high dissolved solids (Krishnaswami et al. 1982). The monitoring results from Georgia also indicated the need for a lower screening level, because the standard deviation of replicate analyses was twice that based on counting statistics (Cline et al. 1983). The EPA is currently conducting a national monitoring program which includes analysis of gross alpha particle activity, ^{226}Ra and ^{228}Ra. It will be very important to evaluate this data set and compare it with existing monitoring results to determine the adequacy of the present screening levels.

Ever since the interim regulations were established, there has been controversy concerning the screening steps used to identify samples which required ^{228}Ra analysis. In the screening procedure, a value of the gross alpha particle activity not in excess of 5 pCi/L is taken to imply a total radium concentration not in excess of 5 pCi/L, and a ^{226}Ra value less than 3 pCi/L is taken to imply a ^{228}Ra value less than 2 pCi/L. In the absence of other information, the $^{228}Ra/^{226}Ra$ activity ratio was expected to be equal to or less than 0.7. However, it has been shown that $^{228}Ra/^{226}Ra$ activity ratios greater than unity are relatively common. The only analysis of the number of radium MCL violations potentially missed using this screening procedure was based on a detailed study of South Carolina. This analysis was used to estimate that 40% to 50% of the violations in coastal states from Texas to New York were being overlooked (King et al. 1982). In Iowa, 18% of the systems found so far to exceed the radium MCL had ^{226}Ra values less than 3 pCi/L (Kriege and Hahne 1982).

The final regulations may contain changes in the monitoring requirements for ^{228}Ra in drinking water. Therefore, new analytical approaches may be needed. There are several options:

a. Selection of one or more analytical techniques for ^{228}Ra that produce accurate results at low cost, thus allowing for an increased number of sampled analyses. There are no known techniques that satisfy these requirements presently.

b. Development of one or more screening procedures for ^{228}Ra or total radium. For example, gross beta particle activity or total radium measurement after selected intervals for ingrowth of radium progeny could be applied if the detection limits were sufficiently low.

c. Requirement for a technique that measures ^{226}Ra and ^{228}Ra simultaneously.

It is realized that even low-cost analyses of every community supply for ^{228}Ra will be expensive. The number of samples requiring a ^{228}Ra analysis could be minimized by a study of the types of aquifers which are likely to have very low ^{228}Ra, regardless of the ^{226}Ra or gross alpha particle activity. Those areas which are most likely to have elevated ^{228}Ra could be required to undergo analyses for ^{228}Ra according to more stringent guidelines. Finally, those areas of unknown ^{228}Ra distribution would be required to follow screening procedures developed from all available data.

III. TOXICOKINETICS

This section will discuss some of the mechanisms of uptake and retention of radionuclides in bone and their possible relevance to radiation toxicity. In general, there are two classes of bone seekers: those that seek bone surfaces and those that seek bone volumes. Plutonium, thorium, yttrium, americium and ^{224}Ra are bone surface seekers, while the alkaline earths, calcium, strontium, barium and radium (226 and 228) are bone volume seekers. Two mechanisms exist for the uptake of radium in the volume of the bone. The first is a surface exchange mechanism, and the second is a sort of self burial mechanism which leads to hot spots. It is known that the first uptake of ^{45}Ca in the bones of dogs and rabbits following injection occurs at bone surfaces, but this ^{45}Ca rapidly returns to the blood (Marshall 1969). Bone formation occurs by apposition of new bone on the existing bone surfaces. Thus, if there is a concentration of radium in the blood and it is high, the bone which is forming will pick up large quantities of this activity, which are often called hot spots. In the first instance, this activity is located at or just under the growing bone surface. However, this radioactivity does not tend to stay in these bone surfaces because it is usually buried by subsequently deposited bone. The new bone contains progressively smaller quantities of the activity, because the specific activity in the blood would be continuously falling unless new radioactivity were ingested.

Because radium is deposited throughout the volume of bone, one would expect that the emitted radiations could reach the red bone marrow. It is this supposition that has led to estimates of leukemias due to ingested radium. There is some controversy about the radiation dose to red bone marrow from radium and the possibility of its leading to leukemias. (Mays, 1983)

There is some evidence, although limited, that the ^{226}Ra concentration in bodies per gram of body ash is constant throughout life especially in bone. That is, the amount of radium in body ash reaches an equilibrium value fairly quickly. This conclusion is usually reached by examining the ratio of ^{226}Ra to calcium in human skeleton, and it appears to be nearly constant with age, starting as early as four months (Wrenn et al. 1983).

A. Model for Radium

The normal intake of radium and the dosimetry of ^{226}Ra and ^{228}Ra have been the subjects of several reviews. Generally, air is an unimportant route of intake for ^{226}Ra or ^{228}Ra, and food is normally the major source of exposure. Water may be the dominant source of intake whenever the concentration of either isotope exceeds several pCi/L. The United Nations

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(a) Adapted from Wrenn et al. 1983.

Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) reviewed our knowledge about normal and elevated intake of Ra in air, water and food and its content in the human body (UNSCEAR 1966, 1969, 1972, 1977, 1982). The ICRP report (1972) on the "Alkaline Earth Metabolism of Man" focuses heavily on the metabolic response to transient intakes, because of the the importance of the occupational and medical radium cases. The dosimetry of radium and other alpha-emitters in bone was summarized by Spiers (1968) in his monograph on internal emitter dosimetry. The discussion here draws heavily on the UNSCEAR summaries.

Although the ratio of ^{226}Ra to calcium in the human skeleton appears to be nearly constant with age, it is not to say that the body does not discriminate against Ra uptake relative to Ca. The observed ratio (OR) in bone and diet is defined as:

$$\text{OR (bone/diet)} = \frac{\text{g Ra/g Ca in bone}}{\text{g Ra/g Ca in diet}} \qquad (1)$$

The OR is considerably less than unity for Ra, which means that Ra is discriminated against for inclusion in the skeleton relative to calcium.

A global mean OR of 0.024 or median of 0.020 for ^{226}Ra is suggested by UNSCEAR in its 1977 survey of the world literature (UNSCEAR 1977). This includes the results of eight studies among six countries, in which the OR ranged from 0.013 to 0.039. The highest single value, 0.039, was found in San Francisco and the second highest U.S. value was 0.024 for New York. In any one study, sampling or other errors which result in a high estimate in bone or a low estimate in diet or the failure to include significant non-dietary sources would lead to an overestimate of OR. Using the global average which falls between the two U.S. values, 0.92 pCi/day intake, and a mean concentration of 5.1 pCi/kg in bone (mass of mineralized bone = 5 kg for Reference Man(a) (ICRP 1975) leads to a net burden in skeleton of 27 days intake equivalent, with a range from 20 to 37 days. Considering the potential variability of the data from which these times are derived, this range seems small indeed.

However, in order to make the best estimates of Ra content in man from dietary information, one ought to know the Ca intake as well as the Ra intake, as was pointed out by Penna-Franca et al. (1965).

For ^{226}Ra, it is possible to measure the body burden in vivo at low normal levels (i.e., about 20 pCi) by use of the breath radon technique, provided that care is taken to have the subject breathe radon-free air for a sufficiently long period. Using the breath radon technique, Stehney and Lucas (1956) measured body burdens of ^{226}Ra and intake rates of

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(a) 5,000 g is mineralized bone, of which 12% is water, 23% is organic and 65% is ash by weight; the dry mass is 440 g (ICRP 1974).

^{226}Ra in the same population. To our knowledge, these are the only cases in which measurements of body burden and intake are available for the same people. Intake of ^{226}Ra was assessed by measuring ^{226}Ra in feces, which should give a good indication of total intake, since only a small fraction of ingested ^{226}Ra is retained in the body for more than a few days. This avoids the necessity of reconstructing representative diets, which introduces errors if foods and water are not included in their proper representative proportions. Processing, in the preparation of coffee, for example, may also alter the Ra content of the water used, and this source of error is avoided by measuring fecal content. Stehney and Lucas (1956) measured the four groups summarized in Table III-1. The Stateville group was from a prison having high ^{226}Ra in water, and presumably, these subjects had had a constant intake since their incarceration. Since that represented a change from low to high ^{226}Ra intake, these subjects were not in equilibrium. The elevated body burdens of ^{226}Ra were not established rapidly, requiring between 10^3 and 10^4 days to approach equilibrium.

The evaluations of "days of intake equivalent body burden" range from 17 for the Stateville group (11 subjects, with a mean time at Stateville of 20 years, probably not in complete equilibrium) to 45 days for eight boys from Lockport, Illinois (ages 15 to 18). With the exception of the latter group, the

Table III-1 Body ^{226}Ra Contents and Daily ^{226}Ra Intakes (Inferred From Fecal Excretion) of Adult and Adolescent Human Males. Their ^{226}Ra Was Accumulated Entirely From Environmental Sources (Food, Water, Air)

Group	No. of subjects	Mean age	No. of fecal samples	Excretion per day[a]		Ratio of body Ra to fecal Ra
				Dry feces (g)	Radium (10^{-12}g)	
Adult control	1	29	3	26.6 ± 0.1	1.7 ± 0.4	24
Stateville	11	44	19	24.1 ± 3.8	12.6 ± 2.4	17[b]
Chicago boys	7	17	26	24.7 ± 1.1	1.6 ± 0.1	22
Lockport boys	8	17	24	37.3 ± 3.2	8.2 ± 0.6	45

[a] Errors shown are the standard deviation of the mean for the group.

[b] Ratio for radium retained by 11 subjects during a 19.7-year average period at Stateville.

Adapted from Stehney and Lucas (1956).

results are consistent with a value of 23 days, which can be derived from the alkaline earth model adopted by ICRP (1972). Further, assuming dietary intakes of 1 g Ca/day, the results are consistent with an OR (bone/diet) of 0.02.

Between 10% and 30% of the Ra in the adult human body has been reported to be in soft tissues. The ICRP model of alkaline earth metabolism suggests that 15% is an appropriate estimate under chronic exposure conditions (ICRP 1973).

For a global average, the days equivalent intake for body ^{226}Ra approaches 27 (UNSCEAR 1977). The ICRP alkaline earth model predicts 23 days, and the data on measured human accumulation and intake rate range from 17 to 45 days (Stehney and Lucas 1956). Since the alkaline earth model includes biological knowledge not inferable from empirical measurements of ^{226}Ra in food and bone, it seems reasonable to pick a value of equilibrium content of radium in the skeleton of about 25 days intake equivalent as a best estimate.

One needs to infer the days equivalent intake for ^{228}Ra and ^{224}Ra; this will be done using parameters adopted by ICRP for the alkaline earth model. Radium-224 is limited by its short half-life of 3.8 days. Radium-228 is ubiquitous in soils, rocks, foods and water, and its fundamental metabolism is the same as that of ^{226}Ra.

If one considers the skeleton to be a single compartment with elimination proportional to content, the equilibrium amount under conditions of chronic intake is proportional to the effective half-time in bone. This half-time is a combination of physical decay and biological removal for ^{228}Ra, but is almost exclusively due to biological removal for ^{226}Ra. The effective half-time of ^{228}Ra in bone should be about 3.7 years, versus 10 years for ^{226}Ra, assuming a single long-term compartment in bone with a biological half-life of 10 years. The relative equilibrium of the two nuclides under conditions of chronic intake should be in the ratio of their effective half-lives or (3.7 years)/10 years = 0.37. Thus, equivalent days intake would be 25 days x 0.37 = 9.2 days. For ^{224}Ra, the following is assumed; 20% GI uptake, 20% transfer blood to bone and a mean life of 5 days in bone, for an equivalent bone burden of 0.2 x 0.2 x 5 = 0.2 day intake. The ^{224}Ra will be distributed on bone surfaces and will have insufficient time before decay to become distributed throughout bone volume. The estimate of the equilibrium content in human skeleton is given in Table III-2.

B. Intake Value for Model Calculations

The fraction of ingested radium that goes to blood (f_1) is reported to be in the range of 0.15 to 0.21 for humans (ICRP 1979). The ICRP 30 report (ICRP 1979) uses the value of 0.2 for all radium compounds. These values are for humans, and

Table III-2 Days Equivalent Intake of Radium Isotopes in Bone

Nuclide	Days equivalent intake	Distribution in bone
^{226}Ra	25.0	Volume
^{228}Ra	9.2	Volume
^{224}Ra	0.2	Surface

The f_1 value for ingested radium is reported to be in the range of 0.15 to 0.21 for humans (ICRP 1979). The ICRP 30 report uses the value of 0.2 for all radium compounds. These values are for humans, and it should be noted that the value for animals is in the range of 0.02 to 0.03 (Stara et al. 1971).

it should be noted that the value for animals is in the range of 0.02 to 0.03 (Stara et al. 1971).

IV. HUMAN EXPOSURE

A. Introduction

At the time of the promulgation of the interim regulations, ^{226}Ra was mentioned specifically because it was felt that this isotope had the most important contribution to human exposure of the naturally occurring radioisotopes. It was expected that radium would be found primarily in groundwaters and only occasionally in surface waters. Furthermore, it was expected that, of the groundwater supplies, radium content would be higher in small systems which relied on fewer wells than in larger systems drawing from large well fields or high-production wells. These expectations were based on the results of an EPA survey which sampled 74 of the larger population centers. Very few supplies were found to exceed a ^{226}Ra concentration of 1 pCi/L (USEPA 1976b). There were few other data available at that time. The U.S. Geological Survey (USGS) compiled data for 509 measurements of ^{226}Ra in groundwater (not necessarily drinking water) for the period between 1954 and 1957 (Scott and Barker 1962). Their results showed that the ^{226}Ra distribution was log-normal, with median values for the ten groupings of geologic provinces sampled ranging from less than 0.1 to 1.0 pCi/L. The USGS later conducted a similar study of existing ^{228}Ra data (Johnson 1971). As early as 1959, it was known that both ^{226}Ra and ^{228}Ra occurred in elevated concentrations in the deep sandstone aquifers of the Midwestern states of Iowa, Illinois, Wisconsin and Missouri (Krause 1959). In this study, it was concluded that the $^{228}Ra/^{226}Ra$ activity ratio was highly variable, and that in order to calculate the total dose, ^{228}Ra should be measured.

Promulgation of the interim regulations instigated state monitoring programs, resulting in detailed studies of areas of elevated natural radioactivity. From these results, much information was gained about the occurrence of ^{226}Ra because of the number of analyses triggered by the gross alpha particle screen (although the exact number of analyses is not known). However, very few data still exist for ^{228}Ra, and it has been necessary to devise other methods to predict its occurrence.

Table IV-1 shows the relative source contribution due to air and food.

B. Compliance Data

1. Surface Water

Surface waters are used by nearly 20% of the community water systems, but serve 66% of the population using community water supplies (Table IV-1). The radium content of surface water is usually very low. Radium-226 generally ranges between 0.1 and 0.5 pCi/L, and the $^{228}Ra/^{226}Ra$ activity ratio is generally greater than unity (Moore 1969, Elsinger et al. 1983).

Table IV-1 Relative Source Contribution to the Daily Intake of Natural Radionuclides (The Drinking Water Contribution Assumes that Two Liters Per Day is Ingested)

Radionuclide	Source	pCi/day	Reference
^{226}Ra	Air	0.007	UNSCEAR (1982)
	Food	1.4	NCRP (1975)
		1.7	NCRP (1984)
		1.1	UNSCEAR (1982)
	Drinking water	Generally small from surface supplies	UNSCEAR (1982)
		0.01-0.2	NCRP (1984)
		0.6-2	Cothern (1985)
^{228}Ra	Air	0.007	UNSCEAR (1982)
	Food	1.1	NCRP (1975)
		1.1	UNSCEAR (1982)
	Drinking water	0.8-2	Cothern (1985)

Also, standard water treatment methods are known to remove radium (USEPA 1976). No surface water violations for radium have been reported by the states. Thus, surface water systems should be addressed separately in the revised regulations. They could be released from monitoring requirements for radium once the source stream was documented as having low natural radioactivity, or the interval for repeat monitoring could be extended.

2. Groundwater

Out of the nearly 60,000 public water supplies in the United States, about 80% use groundwater sources. Over 70% of the groundwater systems serve less than 500 people and are classified as very small (Table IV-2). In general, radium in drinking water is a small-system problem.

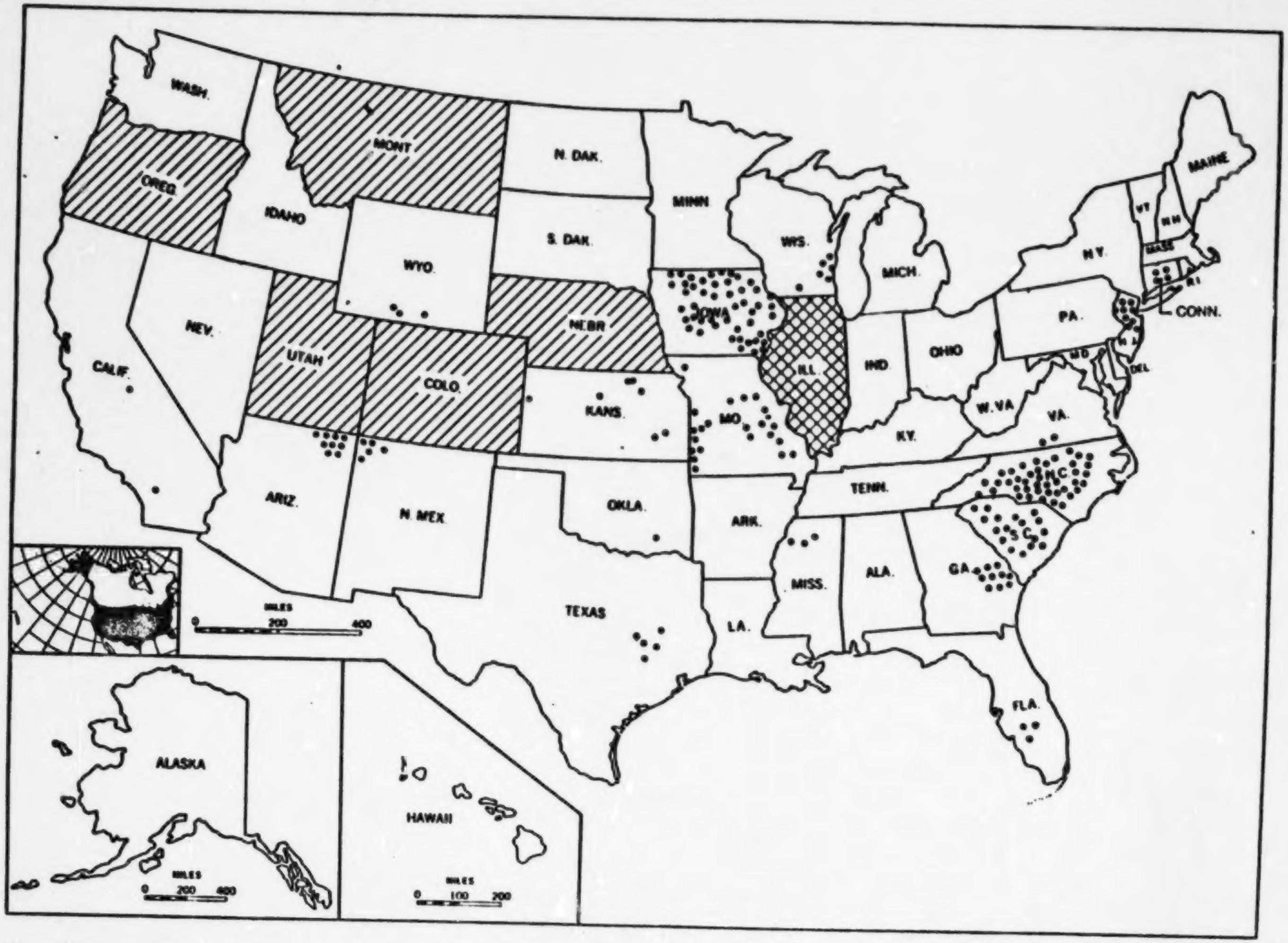
As of 1983, all but six states (Illinois, Nebraska, Colorado, Utah, Montana and Oregon) had reported known MCL violations for radium as required by the interim regulations. There were approximately 200 reported public water suppliers with ^{226}Ra activities in excess of 5 pCi/L after normal treatment (Cothern and Lappenbusch 1983).

The available state compliance data for radium are based almost exclusively on data from samples which first showed a

Table IV-2 Distribution of Public Water Supplies By Source and Population

Type (population)	Surface water		Groundwater		Total number of facilities	Population served (thou.)
	Number of facilities	Population (000)	Number of facilities	Population (000)		
Very small (less than 500)	3,937	776	34,799	4,846	38,736	5,622
Small (500-3,300)	3,817	5,791	10,168	13,221	13,985	19,012
Medium (3,300-10,000)	1,679	9,859	2,233	12,775	3,912	22,634
Large (10,000-100,000)	1,552	46,154	1,203	28,348	2,755	74,502
Very large (greater than 100,000)	217	78,366	55	14,286	272	92,652
TOTALS	11,202	140,946	48,458	73,476	59,660	214,422

Office of Drinking Water, U.S. Environmental Protection Agency, Washington, D.C. 20460


WASH.
OREG.
CALIF.
NEV.
IDAHO
MONT.
WYO.
UTAH
COLO.
ARIZ.
N. MEX.
N. DAK.
S. DAK.
NEBR.
KANS.
OKLA.
TEXAS
MINN.
IOWA
MO.
ARK.
LA.
WIS.
ILL.
MISS.
MICH.
IND.
KY.
TENN.
ALA.
OHIO
W. VA.
VA.
N.C.
S.C.
GA.
FLA.
PA.
N.Y.
VT.
N.H.
MAINE
MASS.
R.I.
CONN.
N.J.
MD.
DEL.
ALASKA
HAWAII
MILES
0
200
400
100

---

[a]The ^{228}Ra was added to the ^{226}Ra when its value was known. The locations are approximate. No data have been reported for those states with diagonal hatching. For the state of Illinois, only gross alpha particle activity has been reported (it would be expected that some radium concentrations will exceed 5 pCi/L).

Figure IV-1(a) Locations of Public Supplies that Exceed 5 pCi/L of Combined Radium[a]

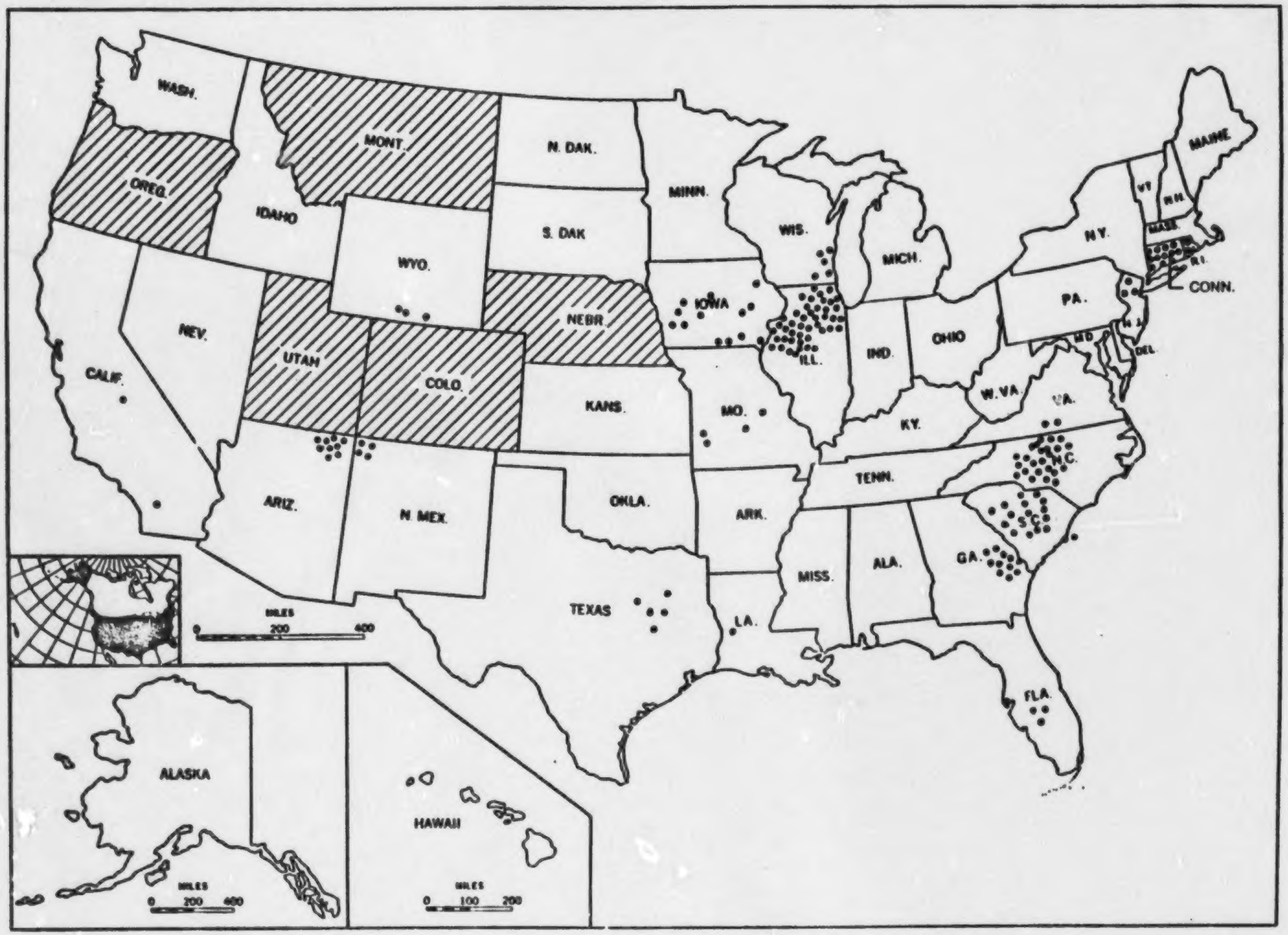

WASH.
OREG.
IDAHO
MONT.
WYO.
N. DAK.
S. DAK
NEBR.
MINN.
WIS.
MICH.
N.Y.
VT
N.H.
MAINE
MASS.
R.I.
CONN.
PA.
N.J.
MD
DEL.
IOWA
ILL.
IND.
OHIO
W. VA.
VA.
N.C.
S.C.
KY.
TENN.
CALIF.
NEV.
UTAH
COLO.
KANS.
MO.
ARIZ.
N. MEX.
OKLA.
ARK.
MISS.
ALA.
GA.
TEXAS
LA.
FLA.
ALASKA
HAWAII
MILES
0
200
400
MILES
0
200
400
MILES
0
100
200

Figure IV-1(b) Locations of Public Supplies that Exceed 15 pCi/L of Gross Alpha Particle Activity[a] gross alpha particle activity of greater than 5 pCi/L. Iowa used a screen of 2 pCi/L for gross alpha particle activity. In some areas, states would analyze additional samples in an area where high radioactivity was found during the initial sampling. Radium-228 data were provided for about one-half of the 200 values reported for ^{226}Ra.

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[a] The uranium and radium activity has been subtracted. The locations are approximate. No data have been reported for those states with diagonal hatching.

From the data reported by the states, the mean total radium activity for supplies exceeding the MCL was almost 10 pCi/L. Radium-226 activity was generally greater than ^{228}Ra activity. Yet, these data were initially biased toward high ^{226}Ra. Figure IV-1 is a compilation of the general areas and specific sites which have high radium concentrations in groundwater as reported from state compliance data. From the available data, there are two specific geological regions where over 75% of the known radium violations occurred:

a. The Piedmont and Coastal Plain provinces in New Jersey, North Carolina, South Carolina and Georgia.

b. A north-central region, consisting of parts of Minnesota, Iowa, Illinois, Missouri and Wisconsin.

The rest of the violations are generally scattered clusters, notably along the Arizona-New Mexico border, Texas, Mississippi, Florida and Connecticut (Figure IV-1). All of these scattered violations had high ^{226}Ra activities, as would be expected from the screening methods used to detect them. Radium-228 activities in these systems were generally very low. It is likely that the current analytical protocol has detected a large percentage of the systems with high ^{226}Ra. Cothern and Lappenbusch (1983) have used the compliance data for ^{226}Ra to estimate that approximately 500 systems will be found to exceed the MCL of 5 pCi/L after the initial monitoring is complete.

Improvement on this estimate is difficult with the existing data base, which is comprised mostly of reported MCL violations for ^{226}Ra. Statistical analysis of these data is not possible because they were not randomly sampled. In this respect, states should be requested to submit all radium results to facilitate further analysis. However, at the National Workshop on Radioactivity in Drinking Water, the following estimates were made to corroborate the previous estimates of MCL violations (Hess et al. 1983). Data from South Carolina (King et al. 1982) showed that approximately 3.0% of the groundwater supplies exceeded the 5.0 pCi/L limit for total radium. Applying that percentage to North Carolina and Georgia, both of which exhibit similar hydrogeological characteristics, produces an estimate of 150 violations for all three states. In Iowa, approximately 10% of the 605 supplies sampled to date (using lower screening criteria) exceeded the MCL. Again, applying this percentage to all the groundwater systems of

Iowa and half of Illinois, Missouri and Wisconsin yields 120 violations for Iowa, 75 violations for Illinois, 50 for Missouri and 60 for Wisconsin. Estimates of violations for the states that have not reported were as follows: ten each for Utah, Colorado and Nebraska and zero for Oregon and Montana. There were 71 violations reported in all other states. The total of these known and estimated violations is 556. Assuming that 10% to 25% of the actual violations were missed during the prescribed screening procedure, the number of violations was estimated to be between 600 and 700 (Hess et al. 1983). However, it should be noted that these estimates are based primarily on ^{226}Ra data. Without additional data, it was not possible to estimate the contribution of elevated ^{228}Ra with the presently used analytical protocol. The number of samples missed by the gross alpha particle activity screen has not been determined. King et al. (1982) found that the average ^{228}Ra/^{226}Ra activity ratio was 1.2 for over 180 samples throughout South Carolina. Of these, ten samples had ^{226}Ra less than 3 pCi/L, but total radium greater than 5 pCi/L. King et al. (1982) estimated that perhaps 40% to 50% of the total radium violations for the Piedmont and Coastal Plain provinces were missed using the prescribed screening procedure which couples ^{228}Ra analysis to ^{226}Ra. In Iowa, 18% of the systems detected so far exceeded the radium MCL, yet had ^{226}Ra less than 3 pCi/L (Kriege and Hahne 1982).

Data on ^{224}Ra activities in groundwater are scarce. However, it appears that the activity of ^{224}Ra may be equal to or up to twice the ^{228}Ra activity (Elsinger 1983) and, therefore, could approach 30 to 40 pCi/L. This ^{224}Ra activity is unsupported; activities of its parent ^{228}Th are usually less than 0.01 pCi/L. Thus, it enters groundwater by alpha recoil during decay of ^{228}Th adsorbed on the surface of aquifer solids. However, the body content of ^{224}Ra and its daughters is small relative to ingestion rates because of their extremely short half-lives and is not believed to be of human health concern (Wrenn et al 1983).

3. Estimating the Occurrence of ^{228}Ra

Most of the available data concerning levels of radium in drinking water are from detailed studies which followed promulgation of the interim regulations. Statewide summaries of ^{226}Ra and ^{228}Ra data have been published for Georgia (Cline et al. 1983), South Carolina (King et al. 1982), Iowa (Kriege and Hahne 1982) and Illinois (Rowland et al. 1977). Lucas (1982) reported results for over 90% of the communities in Illinois, Iowa, Missouri and Wisconsin. One common conclusion of these studies was that a significant number of MCL violations may go undetected using the prescribed screening protocol. In particular, there was a consensus that the coupling of ^{228}Ra with ^{226}Ra was not justified due to the differences in the geochemical behavior of their parent isotopes (Hess et al. 1983). In fact, the analytical scheme could even be biased away from areas of elevated ^{228}Ra. The EPA identified the need for occurrence data on ^{228}Ra for input into the final regulations for both health and economic considerations. However, the existing data on ^{228}Ra were too inadequate to even estimate its distribution in community drinking water so that an analysis could be made of the effect of decoupling ^{228}Ra from ^{226}Ra. Analytical methods for measuring ^{228}Ra in environmental samples are difficult and expensive, which limits the effectiveness of a large-scale monitoring program to actually measure levels of ^{228}Ra in drinking water. Also, the distribution of ^{228}Ra has been shown to be log-normal (King et al. 1982), with most values below the present detection limit of 1 pCi/L, which further limits the effectiveness of a random nationwide monitoring program in identifying new areas of elevated ^{228}Ra. It was realized that without a new approach to determine the occurrence of ^{228}Ra, the human health and economic effects of revising the drinking water regulations for radioactivity would be difficult to evaluate.

In 1982-83, EPA funded two studies to determine the feasibility of using aquifer type and water quality characteristics to predict the occurrence of ^{228}Ra in drinking water (Michel and Pollman 1982, 1983). In these studies, an aquifer classification scheme was developed and tested on three major hydrogeological provinces: the Atlantic Coastal Plain, the

Piedmont and part of the glaciated Central Platform. These regions were selected because of the large existing data base of ^{228}Ra measurements. The aquifer classification scheme was developed from an understanding of the geochemical behavior of ^{228}Ra and its parent ^{232}Th. The studies showed that there were distinct differences in the relative distribution of ^{228}Ra by aquifer type and water quality; a summary of these conclusions is given below.

Aquifers which had low activities of ^{228}Ra included the following:

a. Chemical precipitates (limestone and dolomite) had a log mean ^{228}Ra of 0.3 pCi/L for 120 samples. These rock types have very low thorium content due to their precipitation from water and the very low solubility of thorium.

b. Metamorphic rock aquifers also had a log mean of 0.3 pCi/L for 75 samples. Metamorphism of all degrees tends to recrystallize any loosely bound thorium into resistant minerals, which minimizes the ability of ^{228}Ra to go into solution. Also, groundwater flow in these rock types generally occurs through fractures, rather than through pore spaces, which decreases the rock surface area in contact with water.

c. Quartzose sand and sandstone aquifers generally produced groundwater with low ^{228}Ra, particularly if the total dissolved solids (TDS) were below 1,000 mg/L. The high quartz content of these sediments indicates a high degree of weathering or derivation from a source which had low thorium content. There are two special subclassifications in this class. Sand and gravel aquifers of glacial origin are generally low in ^{228}Ra (log mean of 0.6 pCi/L for 17 samples), regardless of their composition, because of the large size of the clasts and the low surface area to water ratio. When these aquifers are poorly sorted (i.e., contain various amounts of clay, sand and gravel), the clay tends to rapidly absorb radium. Sand and gravel aquifers of alluvial origin also generally have low ^{228}Ra (log mean of 0.7 pCi/L for 56 samples), unless they have large amounts of feldspathic minerals which are high in thorium. Alluvial aquifers are usually composed of quartz-rich sediments and produce large quantities of water.

d. Though not included in the early studies, aquifers composed of basic igneous rocks, such as basalt, are classified as having low ^{228}Ra. Basalts have low thorium content but, more importantly, rapidly adsorb any ^{228}Ra from groundwater. Basalts are such efficient adsorbents that they are being considered as candidate sites for disposal of radioactive wastes.

There are three main aquifer types which produce groundwater with relatively elevated ^{228}Ra activities:

a. Aquifers composed of granitic rocks which have not been metamorphosed have a log mean ^{228}Ra of 1.4 pCi/L and a range of up to 23 pCi/L for 42 samples. Granites are composed of large amounts of coarse-grained feldspar which contain intergranular thorium. Radium-228 produced from this type of thorium distribution is more likely to go into and stay in solution. Igneous rocks of similar composition, but which are fine-grained, such as light-colored volcanic rocks, tend to have lower ^{228}Ra because of rapid readsorption.

b. Arkosic sand and sandstone aquifers have medium to high ^{228}Ra (log mean of 2.4 pCi/L for approximately 70 samples). Arkoses, by definition, contain a significant amount of feldspathic minerals. Arkosic sediments derived from granitic source rocks have an even higher ^{228}Ra. There is a gradient in the ^{228}Ra content of these aquifers, depending upon the amount and composition of feldspar and rock fragments.

c. Quartzose sandstone aquifers with high total dissolved solids were found to have a wide range of elevated

> ^{228}Ra activities from 0.05 to 15 pCi/l. All of these types of aquifers studied were located in the Glaciated Central Platform province and generally had high ^{226}Ra as well. It was concluded that in clean, quartz sandstones with TDS of greater than 1,000 mg/L, competition for the few available sorption sites on the aquifer solids was the controlling factor in keeping ^{228}Ra in solution. This aquifer type may be one of the most overlooked sources of elevated ^{228}Ra, especially in areas where uranium has been removed by groundwater leaching. With the uranium removed, the gross alpha particle screen would be low, and neither ^{226}Ra or ^{228}Ra would be measured.

It is anticipated that an aquifer classification scheme will be used in conjunction with the results of the current EPA nationwide monitoring program to develop an occurrence profile for ^{228}Ra from which population risks and number of systems affected can be determined for various MCLs and analytical requirements for ^{228}Ra in the final drinking water regulations. In addition, it may be possible to include in the regulations a delineation of regions or aquifer types which have different monitoring requirements for ^{228}Ra depending on their risk of having elevated ^{228}Ra concentrations. The interim regulations leave it up to the states to determine the presence of areas of potentially elevated ^{228}Ra and to conduct additional monitoring. A preliminary classification has been made of the entire U.S., by county, of the relative risk of exhibiting elevated ^{228}Ra concentrations. If the distribution of ^{228}Ra for each class can be adequately quantified, then specific monitoring protocols can be developed and applied either as guidelines or requirements.

V. HEALTH EFFECTS IN ANIMALS

Prior to 1944, very little animal experimentation was available concerning the health effects of radium. In general, the numbers of animals investigated were too small, and the doses used were too high. A description of the earlier studies and of the other studies up to 1970 is contained in a report by Chiacchierini et al. (1970).

There have been three large studies of the health effects of radium in animals: two with large mammals, namely beagles, and one with rodents. These three studies will be discussed here. The two mammal experiments involved long-term studies of biological effects of internal emitters and were conducted at the University of Utah (Mays and Finkel 1979) and the University of California at Davis (Raabe et al. 1983). In addition, a rodent study, involving long-term effects of ingestion of radium in mice, was conducted at Argonne National Laboratory (Finkel 1953). The studies of mice and beagle dogs were all done by injection. There are no known studies of low-level feeding experiments for animals.

A. Utah Study

According to Chiacchierini et al. (1970), the Utah experiment was initiated in the early 1950's and was designed to compare the effects of ^{239}Pu and ^{226}Ra. These studies have shown that deaths result from osteosarcoma. A recent paper from these studies (Mays and Finkel, 1979) shows that at the higher levels, bone sarcomas do result from low-dose exposure of beagles to ^{226}Ra. The numbers of dogs injected and of those dying from bone sarcomas are shown in Table V-1. The resulting dose-response curve for beagles in the Utah experiment, as of 1980, is shown in Figure V-1. As can be seen from Figure V-1, these data support the conclusion that a linear relationship between bone sarcoma incidence and dose cannot be rejected by the data in the studies of beagles at Utah.

The group at Utah also investigated the health effects due to injection of radium in mice. Groups of C57BL/Do (black and albino) mice were injected with graded activities of several radionuclides, including ^{226}Ra, and were followed throughout life (Taylor et al. 1983). These studies showed that bone sarcoma was the principal radiation-induced endpoint. As shown in Figure V-2, the dose-response curve from these studies appears to be linear.

B. Davis Study

The University of California study at Davis started in the middle 1950's with the idea of evaluating the relative toxicity of ^{90}Sr and ^{226}Ra in the beagle dog. It was discussed in a preliminary way by Chiacchierini et al. (1970) and has been

Table V-1(a) Bone Sarcomas in Beagles Treated with ^{226}Ra or ^{90}Sr

Nuclide	Inj. (uCi/kg)	Av. yr inj. to death	Treated dogs	Sar. dogs	Incidence (%)	Av. skel. rads 1 yr before death
^{226}Ra	10.4	2.86	10	9	90.0	13400
	3.21	4.13	13	12	92.3	5700
	1.07	6.12	12	11	91.7	2500
	0.339	10.05	13	5	38.5	1100
	0.166	9.40	14	1	7.1	447
	0.062	---	23(3)[a]	2	8.7	210
	0.022	---	25(4)[a]	1	4.0	74
	0.0074	---	10(2)[a]	0	0	25
	0	---	44(14)[a]	0	0	0

[a] Young adult controls for ^{239}Pu, ^{226}Ra, ^{228}Ra, ^{228}Th and ^{90}Sr.

Adapted from Mays and Finkel (1979).

Table V-1(b) Bone Sarcomas in Low Dose ^{226}Ra Beagles (31 March 1979)

Injected (uCi ^{226}Ra/kg)	Injected dogs	Living dogs	Dead dogs	Sarcoma dogs	$\frac{\text{Sar. dogs}}{\text{Dead dogs}}$ (%)	Av. skel. dose 10 yr. post inj. (rad)
0.062	23	3	20	2	10	210
0.022	25	7	18	1	6	74
0.0074	10	2	8	0	0	25
Control[a]	133	35	98	1	1	0

[a] Young adult controls for ^{239}Pu, ^{226}Ra, ^{228}Ra, ^{228}Th and ^{90}Sr.

Adapted from Mays and Finkel (1979).

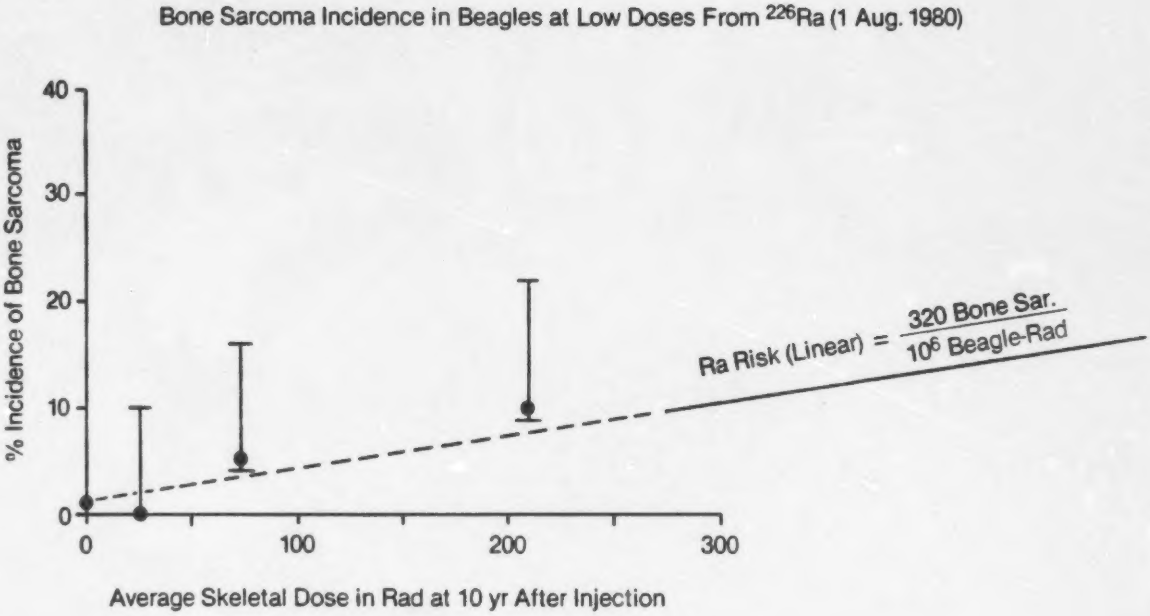


Figure V-1 Bone Sarcoma Incidence in Young Adult Beagles at Low Doses from ^{226}Ra$^{(a)}$

(a) The ratio of bone sarcoma dogs/dead dogs, as of 31 March 1979, is plotted as a solid circle for each dose level. The final incidences, when all of the dogs have died, will be between the indicated upper and lower limits. Shown for comparison is the linear slope from our previously completed study in 51 beagles with radium doses from 183 to 2,500 rad, in which 17 developed bone sarcomas (8). In the on-going low dose radium study, the possibility of a linear dose response cannot be rejected by present data, although the final incidences might give a better fit to an alternative relationship.

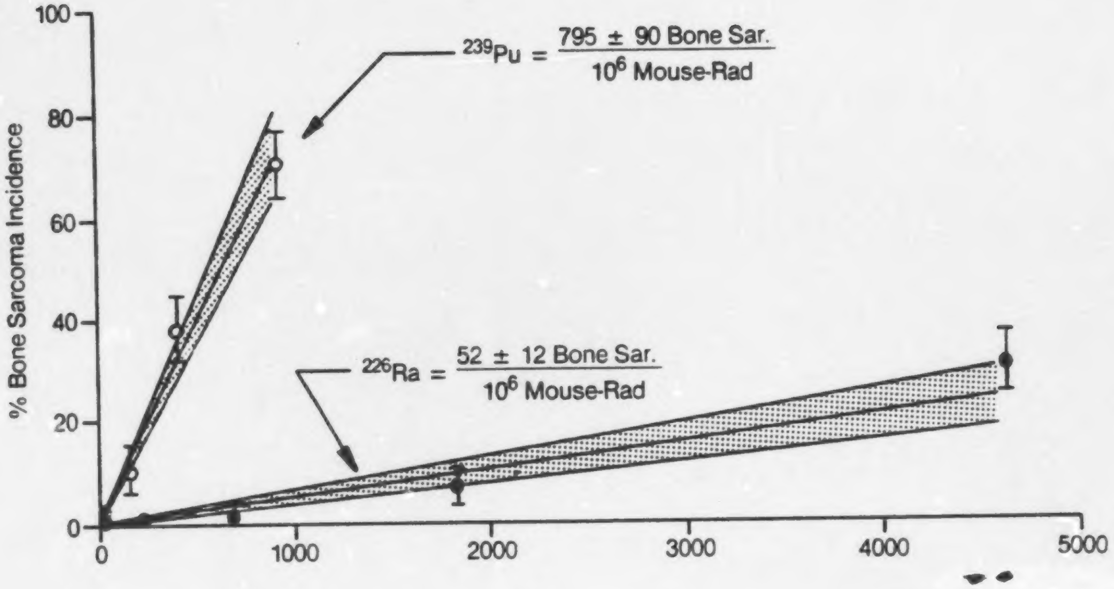


Figure V-2

Plot of % of Bone Sarcoma Incidence as a Function of Dose Comparing Exposure to Ra-226 and Pu-239 for Mice.

described more fully recently (Raabe 1981, 1983, 1984).

The experiment at Davis involved 243 young adult beagle dogs that were given eight fortnightly intravenous injections of ^{226}Ra, starting when they were 435 days of age. These dogs were studied over an 18-year period, and the results, as of June, 1978, are shown in Table V-2. More details on these studies may be found in Raabe et al. (1981).

The researchers from Davis analyzed their data in a three-dimensional form. The three dimensions involved are the two standard dimensions, dose and response, plus a third dimension, time to death. A part of the relationship between dose and time to death is shown in Figure V-3, from Raabe et al. (1981). This figure shows the relationship between mice, dogs and humans. As can be seen, as the dose decreases, the time to death increases in all cases. Eventually, the time to death exceeds the lifetime of the animal or human involved. This crossover point has been suggested as a practical threshold.

C. Argonne Study

As discussed by Chiacchierini et al. (1970), some work was done at Argonne National Laboratory by M. Finkel (1953). This work was done with CF_1 mice. Some results from this study are shown in Table V-3 (Mays 1979). These data are shown in plot

Table V-2 Description of ^{226}Ra Toxicity Studies in Davis Beagles and Status to June 1978 (Eight Fortnightly Injections Started at 435 Days of Age), Giving Total Injected Quantity of ^{226}Ra per Dog in Each Dose Level

Treatment code	Number of dogs	Total injected ^{226}Ra (uCi/kg)	Number alive (6/78)	Bone tumor deaths
R00	78	0	54	0
R05	45	0.024	32	0
R10	36	0.064	24	2
R20	41	0.376	14	18
R30	39	1.12	0	31
R40	41	3.36	0	38
R50	41	10.0	0	27

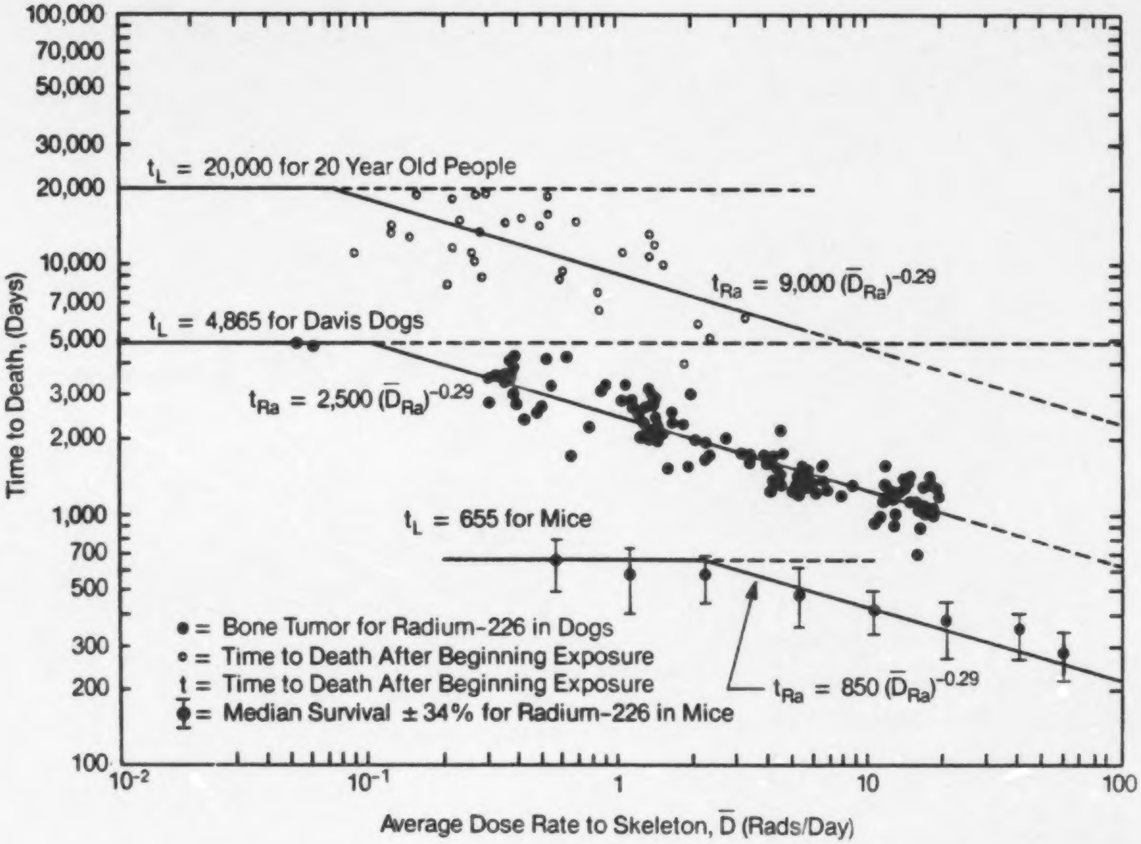


Figure V-3. Two-Dimensional Presentation of Primary Bone Tumor Deaths from ^{226}Ra for People, Davis Beagles and Female CF_1 Mice(a)

form in Figure V-4. As seen from Figure V-4, the dose-response curve shows a slight concave curvature. This curvature is sometimes called superlinear and has been indicated in other studies (NAS 1980).

(a) Similar dose-response functions are indicated by parallel lines with negative slope representing the medians of the respective bone cancer death distributions. Also shown by horizontal parallel lines are median times, T_L of the occurrence of old-age related spontaneous deaths.

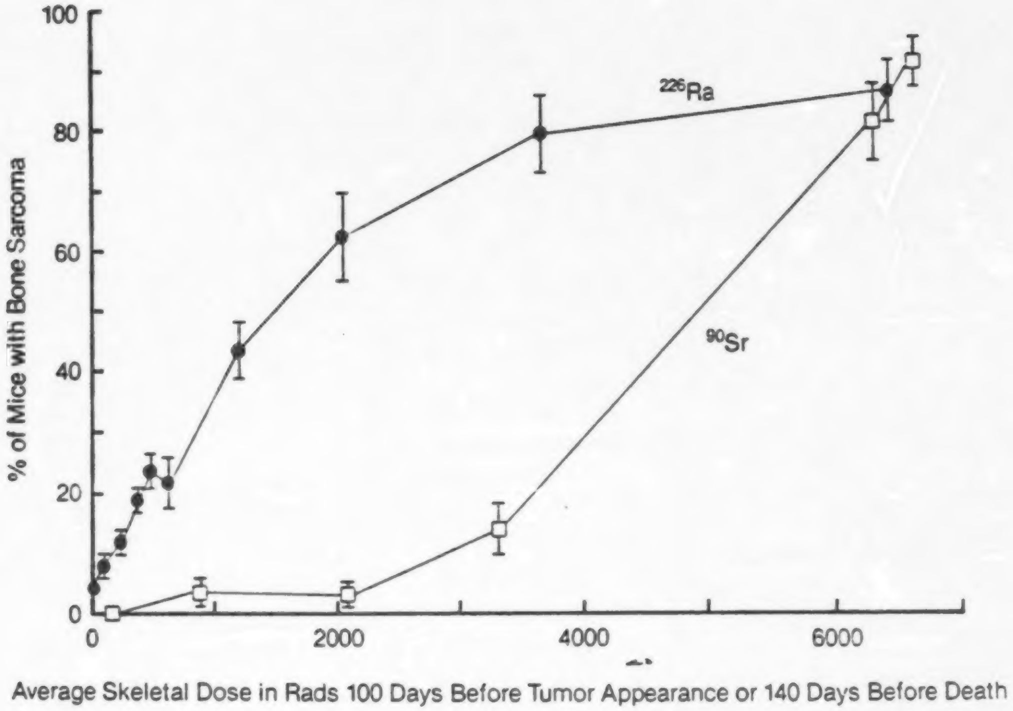
D. Conclusions and Recommendations

The studies on health effects in animals reviewed in this section all indicate that there is a definite relationship between the level of ingested radium and bone cancer. The questions that arise from this research are whether the dose-response curve is linear in the region where the studies were done, whether it is linear at lower doses and whether a threshold exists. Although the Davis group has suggested that a practical threshold exists, it appears that at this time, there is no strong evidence for the existence or non-existence of a threshold in the dose-response curve at this time.

For regulatory purposes, it seems reasonable at this time to assume that these data are consistent with a linear non-threshold model. This conclusion is not proof that the linear non-threshold model is, in fact, the valid one. In the words of the Chiacchierini et al. (1970) report, "It is merely the

Table V-3 Bone Sarcomas in Female CF_1 Mice Treated with ^{226}Ra or ^{90}Sr

					Bone Sarcoma Mice	
	Inj. (uCi/kg)	Mice at 150 days post inj.	Sar. mice	Inc. (%)	Av. days, inj. to appear. (Ra) or death (Sr)	Av. skeletal rads 100 days before appear. or 140 days before death
^{226}Ra	120	45	14	31.1	328	28900
	80	44	31	70.5	359	21300
	40	45	33	73.3	394	11800
	20	44	38	86.4	428	6420
	10	43	34	79.1	484	3640
	5	45	28	62.2	544	2040
	2.5	104	45	43.3	639	1190
	1.25	104	22	21.2	657	614
	1.00	239	56	23.4	643	480
	0.75	504	94	18.7	686	383
	0.50	683	80	11.7	655	244
	0.25	247	19	7.7	580	109
	0.10	252	5	2.0	853	62
	0.05	254	11	4.3	710	26
	0	521	6	1.2	730	0



Adapted from Mays (1979).

Figure V-4 Bone Sarcomas in Female CF1 Mice Injected with ^{226}Ra or ^{90}Sr[a]

most cautious and reasonable approach to assume that any radiation exposure has an accompanying biological effect in the absence of any proof to the contrary." Thus, based on animal studies with beagles and mice, the linear non-threshold model would be the prudent one to use for regulatory purposes.

VI. HEALTH EFFECTS IN HUMANS

An EPA estimate of the body burden resulting from ingestion of radium which was used to develop the existing MCLs for radium was that of the Federal Radiation Council (FRC 1961); i.e., that the skeletal body burden of radium is 50 times the daily radium intake. This value is listed in Table VI-1 along with other parameters.

Although the human health effects due to the ingestion of radium can now be quantified, and dose-response curves can be drawn, the first reported health effects were non-stochastic (Schlenker 1984).

The interim regulations for radioactivity in drinking water were developed using the ICRP 2 model, in which a body burden of 100,000 pCi produces an average bone dose of 30 rem/year. Assuming a direct proportion, 500 pCi would give an average bone dose of 150 mrem/year, as shown in Table VI-1. From the FRC estimate mentioned above, a burden of 500 pCi would be caused by an intake of 10 pCi/day. Thus, an ingestion rate of 10 pCi/day (water concentration of 5 pCi/L) produces a bone dose of 150 mrem/year. At the time that the MCL for radium was promulgated (40 CFR 141, August 14, 1975), the quality factor for alpha particles was ten(ICRP , 1959)

The predicted risk estimates from several sources are shown

Table VI-1 Risk, Dose and Body Burden Estimates From Ingesting ^{226}Ra

Intake (pCi/L)	Body burden (microCi)	Bone dose (mrem/year)	Number of excess fatal cancers[a]/ 10^5 exposed/ lifetime	References
5	0.0005	-		FRC (1961)
5	0.0005	150	5-21[b]	USEPA (1976)
5	-	92	4.4	Sullivan et al. (1981)
5	-	150	2.0	BEIR III (NAS 1980, Appendix A)
-	-	150	0.3-0.8[c]	USNRC (1978)
5	-	80	-	Rowland et al. (1978)
30	0.001	60	-	Evans (1975)
5	-	-	3.4	Rowland et al. (1981)

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[a] Note that the "normal" or background risk is 10^{-5} bone carcinomas/ person/year or, in the units of this column, roughly 70 excess fatal bone cancers/10^{-5} exposed/lifetime (Rowland 1978). It is assumed that most of the cancers involved will be fatal.

[b] 0.7 to 3 excess cancers/10^6 people exposed/year x $\frac{70.7 \text{ year}}{\text{lifetime}}$ = 5 to 21 excess cancers/10^5 people exposed/lifetime.

[c] 0.15 to 0.4 bone cancers/10^6 exposed/rem. This included a quality factor of Q = 10.

in Table VI-1. The range of 0.7 to 3 excess cancer deaths/ million people/year exposed over a lifetime (or 5 to 21 excess cancers/10^5 people exposed/lifetime) was determined for EPA's interim regulation using the data from the BEIR I report (NAS 1972). It was assumed that almost all the cancers would be fatal. The range shown is due to use of the relative and absolute risk models.

In Table VI-1, for an intake of water containing 5 pCi/l, the estimate of 4.4 excess fatal cancers/ 10^5 people exposed/ lifetime is based on recent calculations produced by EPA/Office of Radiation Programs (ORP) (Sullivan et al. 1981); these include other competing risks and the ICRP 30 model. The calculations use the life table approach (Bunger et al. 1981) and an overall risk value of 200 excess cancer deaths/million people exposed/rem. This risk estimate is from a combination of absolute and relative risk estimates. For ingestion of water containing 5 pCi/l the dose-equivalent rate is 83 mrem/year for endosteal bone and 8.6 mrem/year for bone marrow. These dose equivalent rates are calculated to 70 years of age due to a lifetime ingestion of 10 pCi/day. The corresponding risk estimates of fatalities for a lifetime exposure per million people exposed are 8.5 for endosteal bone, 30.8 for bone marrow and 5.1 for other organs.

The recent BEIR III report (NAS 1980) estimates a provisional risk rate coefficient based on combining information from Rowland et al. (1978) and Mays and Finkel (1979) for radiation-induced bone cancer. The coefficient for a linear dose-response curve is 10^{-6} sarcomas/year/person/rad. Comparing the BEIR III linear risk estimate to the other lifetime estimates in Table VI-1 requires the following information: average lifetime is 70.7 years, and ingestion of 1 pCi/year leads to a bone dose of 1.13×10^{-3} mrad/year (Sullivan et al. 1981). From the latter value, ingestion of 2 L/day of water containing 5 pCi of $^{226}Ra/L$ gives a dose of $1.13 \times 10^{-3} \times 365 \times 5 \times 2 = 4.12$ mrad/year. Observing that this is an admittedly simple calculation that can only yield a rough approximation for comparison, the following calculation can be done:

$$\left[\frac{\text{1 sarcoma/year}}{10^{6}\text{ people exposed/rad}}\right]\left[\text{4.12 mrad/year}\right]\left[\text{70.7 year}\right]$$

$$\left[\frac{\text{70.7 year}}{\text{lifetime}}\right] = \frac{\text{20 sarcomas/lifetime}}{10^{6}\text{ people exposed}}$$

Rowland et al. (1981) fit linear and quadratic functions with a cell killing factor to the data on radium watch dial painters. They had data for 1,468 women showing 42 cases of bone sarcoma.

The general expression used was:

$$I = (a + bD + cD^{2}) \exp(-eD)$$

where I denotes bone sarcomas per person-year; D is the quantity, in microcuries, that enters the blood; a is the incidence in the absence of radiation and b, c and e are parameters used to fit the data. We will assume that water containing 5 pCi/L is consumed at the rate of 2 L/day.

$$D = \frac{5\ \text{pCi}}{\text{L}} \times \frac{2\ \text{L}}{\text{day}} \times \frac{365\ \text{day}}{\text{year}} \times \frac{1}{5} = 730 \times 10^{-6}\ \text{microcuries per year}$$

In this expression, 1/5 represents the fraction ingested that enters the blood. For low-dose situations, it is possible that a linear dose-response curve is the actual situation. Using the above value of $D = 730 \times 10^{-6}$ microcuries/year and the value of 1.3×10^{-5} suggested by Rowland (1981) for b yields 1×10^{-8} sarcomas/person-year during the remaining life. Each year of ingestion adds 1×10^{-8} sarcomas/year, but for a shorter period. Assuming a latency period of 5 years and using the admittedly rough approximation that about half the maximum response (reached at 110 years) is reached after 70.7 years:

$$1 \times 10^{-8} \times \left(\frac{110 - 5}{2}\right) \times (70.7 - 5) = 3.4 \times 10^{-5}\ \text{sarcomas/lifetime}$$

From analysis of the New Jersey radium dial painters (Sharpe 1974), there were only 42 cases studied among which 24 had malignant tumors or blood dyscrasias. This rate was higher than the expected normal incidence and greater than that among the siblings or parents. Of these 42 cases, only 31 had body burden estimates, and they may not constitute a valid statistical sample of the whole population. Although the numbers are quite small, the data do suggest that the ingested radium is correlated with paranasal sinus and bronchi malignancies (see Table VI-2, from Sharpe 1974). Other malignancies that might be correlated with the radium ingestion are multiple myeloma and leukemia. In a more recent analysis of the same group, Sharpe (1981) saw radium osteitis or bone infarcts at body burdens as low as 0.0042 microcuries. This is the first evidence of a deleterious effect for body burdens below 0.1 microcuries. The main problems with the NJ radium dial painter studies are the very small number of subjects, which makes statistical inferences dubious, the biases in the data and the 30 or 50 cases for which body burdens cannot be estimated. Because of these difficulties, any conclusions must be questioned. The data, however, are not inconsistent with a linear dose-response curve, and the risk estimate using the linear curve is virtually the same as that estimated by EPA for a daily ingestion of 10 pCi of ^{226}Ra.

A. Shape of the Dose-Response Curve

The shape of the dose-response curve is one of the most important components of the estimates of dose and risk due to the ingestion of radium. The three general possible shapes for this curve are linear, convex and concave. The concave downward slope is sometimes described as convex or decreasing slope with increasing dose. The concave upward slope is where slope increases with increasing dose. The quadratic slope is thus concave upwards, although this is sometimes described as

Table VI-2 Observed Distribution of Malignancies in 42 Autopsied Cases Over Twenty-Five Years

Primary site	Cases (r)	U.S. deaths per 100,000/year from death certificates	
		1950	1960
Paranasal sinuses	4	---	0.8[a]
Bronchi	4	5.1	10.2
Uterine cervix	3	5.5	4.7
Buccal cavity and pharynx	2	3.4	3.4
Rectum	2	6.9	6.1
Multiple myeloma	2	---	---
Osteogenic sarcoma	1	---	1.5[b]
Ovary	1	3.8	4.5
Myelofibrosis	1	---	---
Chronic myelocytic leukemia	1	5.9	7.1
Hodgkin's disease	1	1.7	1.7
Reticulum cell sarcoma	1	2.3	3.3
Pancreas	1	5.9	7.6
Hepatoma	1	---	1.1
Urinary bladder	1	4.2	4.4
Prostate	1	7.5	8.1
Glioblastoma multiforme	1	---	---

[a] An estimate, probably much too high.
[b] An estimate for ages 45 to 69.

Adapted from Sharpe (1974).

concave. As pointed out by Mays (1979), UNSCEAR (1977) and BEIR III (NAS 1980), there is generally support for each of these curves from different experimental evidence. The concave upward curve is supported by the studies of radium dial painters, who ingested both ^{226}Ra and ^{228}Ra, and by studies of mice which ingested ^{228}Ra. The concave downward curve is supported by studies of mice which ingested ^{224}Ra. The linear curve appears to be supported by studies involving the ingestion of other bone-seeking isotopes, including ^{224}Ra in humans, ^{228}Th in beagles, ^{239}Pu in rats and mice and ^{227}Th in mice.

Recently, several questions have been raised regarding the dosimetry of the Japanese A-bomb survivors (Loewe and Mendelsohn 1981). Because of these questions, a program has been started to recalculate the doses received. A gathering of scientists discussed these matters and concluded that the dose estimates that have been in use since 1965 can no longer be considered accurate and should be revised in light of this recent work (Loewe and Mendelsohn 1981). One of the main early conclusions from their work is that the neutron dose had a smaller impact than was previously thought. In addition, they concluded that neutron doses of the two cities were similar and not as different as was previously thought. Thus, it may be possible to combine the data from the two cities and improve the statistical accuracy. It is possible that the risk figures for estimating cancer deaths will change by a factor of two, but it is too early to know exactly what the outcome of the work will be. Because of the question raised concerning the dosimetry for the A-bomb survivors, much of the analysis in the BEIR III report (NAS 1980) is in doubt. In the BEIR III report, some mention is made of high linear energy transfer (LET) radiation. The other places where high (LET) radiation is mentioned appear to be for neutrons, and these may be incorrect due to the errors in dosimetry for the A-bomb survivors.

An attempt to fit 12 studies of the effects of alpha-emitters on humans, dogs and mice with a linear function yielded a range of slopes that differ by more than two orders of magnitude (Mays and Lloyd 1972). The slopes were expressed as percent incidence per rad and were 0.0046, 0.009, 0.043, 0.13, 0.15 and 1.1. A recent paper (Mays et al. 1980) observes that a linear relationship between bone sarcoma incidence and dose in beagles cannot be rejected from present data. The slopes range over two orders of magnitude. This demonstrates the problem of finding a universal fit.

The relative biological effectiveness (RBE) of alpha particles at low dose rates has been recorded to be as high as 26 (Mays and Finkel 1980). The RBE is the ratio of the dose, in rads to produce a given effect with x or gamma rays to the dose, in rads, to produce the same effect with another radiation under investigation. In biology, the quality factor (Q) of the health physicist is similar to the quantity RBE (Grosch and Hopwood 1979). Epidemiological and experimental data suggest that the effect per dose of alpha radiation at low dose rates is greater than that of high dose rates (NAS 1980). Thus, for alpha particles, the new quality factor of 20 (ICRP 1979) may be different low at the 100-mrem/year dose equivalent level.

Because cancer is so poorly understood, very few models of how it occurs exist. However, a theoretical model has been developed to describe how alpha radiation causes bone cancer (Marshall and Groer 1977). In this model, the process starts when an endosteal cell is struck twice by alpha radiation (a two-hit process). The tissue then naturally starts remodeling, and this initiates the first cell of an osteosarcoma. Cells are killed by alpha radiation during this process, and some are replaced by stem cells. The osteosarcoma cell multiplies and becomes observable in two to three years in dogs and in three to six years in humans. Essentially, two alpha particles transform an endosteal cell into a dormant malignant cell. Promotion then proceed because of natural remodeling of the bone. This model fits the observed data from the studies of radium watch dial painters and beagles. It correctly predicts the plateau at high dose rates and a dosesquared tumor rate at low doses. It also predicts that ^{228}Ra is 2.5 times more radiotoxic than ^{226}Ra. However, the the uncertainties in the data are large, and that the theory fits the data does not necessarily prove that the postulates are correct. In particular, this theory does not conclusively demonstrate that the dose-response curve is quadratic in shape or that a two-hit process is really the correct one, especially at low doses.

In a recent paper, Marshall and Pagnamenta (1981) suggest that below 40 rads, the doublet model is promising. In this model, an alpha particle ionizes two DNA bases in the same double helix. This mechanism is consistent with a linear dose-response curve.

Much controversy surrounds the question of whether the dose-response curves for radioactivity are linear or curvilinear. In its BEIR III report, NAS (1980) concluded that there are a number of possible dose-response functions, but that there is no compelling evidence for the validity of any one. In an earlier review, Chiacchierini et al. (1970) concluded that at that time, the information was "not significant to specify an unequivocal dose-response model." The current state of knowledge appears to be no closer to determining that shape.

The convex shape is supported by only two studies: the ^{224}Ra study in mice and the A-bomb study. However, it appears that when ^{224}Ra is ingested, it behaves differently than ^{226}Ra or ^{228}Ra. For example, ^{224}Ra is a bone surface seeker, whereas ^{226}Ra and ^{228}Ra are bone volume seekers (NAS 1980). Also, the appearance time of the bone sarcomas resulting from ^{224}Ra ingestion is shorter (4 to 27 years) than that resulting from ^{226}Ra ingestion, which is as long as 52 years (NAS 1980). This would suggest that ^{224}Ra is a special case, and unless more direct evidence becomes available to explain why ^{224}Ra and ^{226}Ra affect the human body in such different ways, the setting of a radium standard should rest primarily on the experience with ^{226}Ra and ^{228}Ra. One other researcher has suggested that the dose-response curve should be convex (Baum 1973). This work, however, was based on data from the A-bomb survivors, and thus is suspect, due to the errors found in the related dosimetry.

The BEIR III report (NAS 1980, Appendix A) concluded: "It is difficult, on the basis of both experimental and theoretical consideration, to rule out a linear component to the dose-effect relationship for bone-sarcoma induction by alpha-emitters. This component should become dominant at very low doses."

B. Types of Cancer Induced By Radium Isotopes in Man

(This section is taken from Mays, 1983)

At low and medium doses of internally deposited radium, the most severe biological damage is cancer arising from skeletal tissue. For ^{226}Ra, the two types of malignancy are induced: (1) bone sarcomas (mostly osteosarcomas, fibrosarcomas and chondrosarcomas) and (2) head carcinomas (carcinomas of the paranasal sinuses and the mastoid air cells). Among some 3,700 persons in the U.S. who were exposed to ^{226}Ra and ^{228}Ra

by dial painting, medical administration and other means, a total of 85 cases of bone sarcoma and 36 cases of head carcinoma had been observed as of 31 December 1982 (Gustafson and Stehney 1983). However, *no* head carcinomas have yet been observed in the follow-up of 2,324 German patients injected with ^{224}Ra, although 55 have developed bone sarcomas (Mays and Spies 1984, Wick and Gossner 1983). Similarly, no head carcinomas have been observed in persons internally contaminated with ^{228}Ra, unless the dose from ^{226}Ra was also high (Evans 1966, Rowland et al. 1978). This suggests that when ^{226}Ra decays to ^{222}Rn within the body, the accumulation of ^{222}Rn gas in the head cavities is the major inducer of these carcinomas. For pure ^{224}Ra and ^{228}Ra, which do not produce ^{222}Rn gas, the risk from head carcinomas is regarded as trivial compared to the risk from bone sarcomas.

For alpha-emitters deposited in mineral bone, the risk from radiation-induced leukemia in humans has been insignificant relative to that from bone sarcoma. Among the 2,940 males and females located who were radium dial workers before 1970, there were 63 cases of bone sarcoma observed, versus about one case expected naturally, in contrast to the ten cases of leukemia observed,versus the 9.24 cases expected based on natural rates that were age-, sex- and calendar year-specific (Spiers et al. 1983, Rowland et al. 1983). Furthermore, four of the ten leukemias observed were chronic lymphocytic leukemia, a type which has not been shown to be inducible by radiation.

The lifespan risk coefficient for leukemia-induction by alpha particles in humans is very much lower than that obtained by multiplying the ICRP risk coefficient for sparsely ionizing radiation by a quality factor of 20 (ICRP 1977).

$$\begin{array}{l}\text{ICRP risk}\\ \text{coefficient}\\ \text{for alpha}\\ \text{particles}\end{array} = \left[\frac{20\ \text{leukemias}}{10^6\ \text{person}\cdot\text{rem}}\right]\left[\frac{20\ \text{rem}}{\text{rad}}\right] = \frac{400\ \text{leukemias}}{10^6\ \text{person}\cdot\text{rad}} \tag{1}$$

Spiers et al. (1983) have calculated the alpha particle dose to red marrow in a special subgroup of 693 women who were dial workers before 1930, who were measured while living, survived without bone sarcoma for two years after their first measurement, and were alive in 1957. Their alpha particle dose to red marrow averaged about 25 rads. Assuming a similar average dose to the total measured and unmeasured group of 1,285 women who were dial workers prior to 1930, the ICRP risk coefficient would predict about 13 cases of radiationinduced leukemia among these 1,285 women.

$$\left[1{,}285\ \text{persons}\right]\left[25\ \text{rad}\right]\left[\frac{400\ \text{leukemias}}{10^6\ \text{person}\cdot\text{rad}}\right] = 13\ \text{leukemias} \tag{2}$$

The total of predicted leukemias would then have been about 18 (13 radiation-induced plus 5.4 naturally expected). However, the prediction of 18 leukemias is strongly rejected by the observation of only four leukemias among these 1,285 women. If the risk coefficient were ten-fold lower, the predicted total of about 6.7 leukemias (1.3 radiation-induced plus 5.4 naturally expected) would be in reasonable agreement with the four observed leukemias.

Independent evidence that the ICRP risk coefficient for alpha particle-induced leukemia is about ten times too high comes from Robin Mole's (1978) analysis of leukemias in European Thorotrast patients. There were 44 leukemias observed among 4,000 Thorotrast patients who were injected with an average of 25 mL of Thorotrast, for which the dose rate to red marrow was 9 rad/year, giving a dose of 270 rads in 30 years.

$$\begin{array}{l}\text{Leukemia risk}\\ \text{coefficient for}\\ \text{Thorotrast}\\ \text{alpha particles}\end{array} = \frac{44 \text{ leukemias}}{(4{,}000 \text{ persons})(270 \text{ rad})} = \frac{40 \text{ leukemias}}{10^6 \text{ person}\cdot\text{rad}} \qquad (3)$$

The low effectiveness of alpha particles in the induction of human leukemia might be due to the following factors, acting singly or in combination: (1) non-uniform irradiation which allows undamaged stem cells to replace cells killed by irradiation, (2) low frequency of transformable cells in the regions most heavily irradiated by alpha particles, such as regions near the bone surfaces or near the phagocytic cells that take up colloidal Thorotrast, (3) an RBE of less than 20 for uniform alpha particle versus sparsely ionizing radiation at low dose rates for leukemia induction, (4) a lifespan risk coefficient of less than 20 leukemias/10^6 person·rem for sparsely ionizing radiation at low dose rates or (5) other factors. Regardless of which theoretical possibility is correct, by direct observation of humans exposed to graded levels of alpha radiation emitted from mineral bone,it has been established that the risk from induced leukemia is very small compared to the risk from induced bone sarcomas (Spiers et al. 1983, Mays et al. 1983). We have given considerable attention to the risk from leukemia, primarily because a recent calculation suggested that the lifetime ingestion of ^{226}Ra might induce more leukemias than bone cancers (Cothern 1983). However, the opposite situation is indicated by actual observations on the effects of radium isotopes in humans (Spier 1983; Rowland et al. 1983, Mays et al. 1983).

Of the 1,285 women who were dial workers prior to 1930, multiple myeloma has occurred in six, as compared to the 2.1 naturally expected cases (Stebbings et al. 1984). While the three-fold increase is statistically significant ($P = 0.042$), it is not necessarily due to internally deposited ^{226}Ra and ^{228}Ra. The external x-ray exposure from the radium paint pots has been estimated at 8 to 20 rad/year (Stebbings 1984). Of the six women with myeloma, four worked as dial painters for at least 50 weeks, one worked only two weeks, and the duration of employment for the remaining one is unknown (Stebbings 1984). Even if the four excess cases of myeloma were caused by skeletally deposited radium, induced myeloma would be only 6% as frequent as the 63 cases of bone sarcoma observed among these 1,285 women. Among the 2,324 German ^{224}Ra patients, the one case of multiple myeloma (plasmacytoma) was only 2% as frequent as the 55 cases of bone sarcoma observed among these patients (Mays and Spiess 1984, Wick and Gossner 1983). Multiple myeloma in human bone appears to be only slightly inducible by radium.

For the 1,285 women who were dial workers before 1930, Stebbings et al. (1984) have compared the number of observed versus expected cancers for the ten following sites of highest naturally expected frequency: breast, colon, lung, stomach, pancreas, cervix uterus, corpus uterus, rectum, red bone marrow and liver. For none of these cancers was the observed incidence significantly different ($P < 0.05$) from the natural expectation based on rates in the relevant counties. Summing the ten sites, there were 101 observed cancer cases versus the 94 cases expected based on county rates.

It is clear that the 63 bone sarcomas and 23 head carcinomas observed represent the vast majority of radium-induced cancers among the 1,285 women who were radium dial workers prior to 1930, although a small number of additional cancers might possibly have been induced in other tissues (Finkel et al. 1969, Stebbings et al. 1984).

1. Dose-Response Relationships

a. Head carcinomas - The incidence rate for head carcinomas versus ^{226}Ra intake to blood was fitted to a variety of dose-response relationships for the measured female dial workers employed before 1930 (Rowland et al. 1978). A minimal latent period of ten years was assumed. The best statistical fit (P = 0.86) was to the following linear relationship (Rowland et al. 1978):

$$\underset{\text{(carcinomas/person·year)}}{\text{Yearly incidence}} = 1.6 \times 10^{-5}\, \underline{D} \tag{4}$$

where the radium intake to blood is $\underline{D}$ microcuries ^{226}Ra. The intake of ^{226}Ra to blood was back-calculated from the measured body content of ^{226}Ra at "t" days after intake using the Norris retention function (No. 55), $R = 0.54\, t^{-0.52}$ from Norris et al. (1955). The intake of ^{228}Ra was disregarded, since head carcinomas have not been associated with ^{228}Ra.

b. Bone sarcomas - Two groupings were made for analysis of bone sarcoma incidence in the measured female dial workers employed before 1950 (Rowland et al. 1983).

Group A consisted of all measured female dial workers. Their years at risk were assumed to begin five years after the start of employment (allowing for a five-year minimal latent period for bone sarcoma) and to continue until death, the diagnosis of a bone sarcoma or the end of follow-up. Group A contained 42 bone sarcomas among 1,468 persons averaging 40.0 years at risk. It may under-represent persons who died early and were not measured for radium, but over-represent those who ultimately suffered from the effects of radium and were identified and measured as a consequence.

Subgroup B, a part of Group A, was selected to eliminate persons who were possibly measured for radium as a consequence of their cancers. It included only the female dial workers who were measured and excluded any person who died or developed a bone sarcoma within two years after the first measurement. Thus, the years of monitored risk for Subgroup B started two years after the first measurement and continued until death, the diagnosis of a bone sarcoma or the end of follow-up. Subgroup B contained 13 bone sarcomas among 1,257 persons averaging 9.4 years at monitored risk.

For Group A, the incidence rate for bone sarcomas was best fit (P = 0.73) by the following dose-squared x exponential equation (Rowland et al. 1983):

$$\underset{\text{(sarcomas/person·year)}}{\text{Yearly incidence}} = 7.0 \times 10^{-8} D^2 e^{-0.0011 D} \qquad (5)$$

where for bone sarcomas, $\underline{D}$ is the intake to blood in microcuries of ^{226}Ra plus 2.5 times the intake in microcuries of ^{228}Ra, since 1 microcurie ^{228}Ra with high retention of daughters is equivalent for bone sarcoma induction to about 2.5 microcuries of ^{226}Ra with low retention of progeny(Rowland et al, 1978).

No bone sarcomas have yet been identified among any of the exposed measured persons with radium intakes to blood below 50 microcuries (Rowland et al. 1983). Among the 1,321 measured female dial workers with intakes below 50 microcuries, the preceding equation plus the natural incidence predict 0.67 total expected bone sarcomas (0.29 induced plus 0.38 natural). For an expectation of 0.67 sarcomas, zero sarcomas could occur with a 51% probability (P = 0.51).

At low values of intake, the exponential in Equation 5 approaches unity, and the calculated yearly incidence from ^{226}Ra varies as the square of the intake. For a 1 microcurie ^{226}Ra intake, the calculated yearly incidence of 7×10^{-8} is only 1% of the natural yearly incidence of 0.7×10^{-5}. At lower doses, the calculated yearly incidence from ^{226}Ra rapidly approaches zero. We will use zero risk as a lower limit for radium-induced bone sarcomas at low intakes.

For the more epidemiologically suitable Subgroup B, acceptable fits were observed for the following equations (Rowland et al. 1983):

Linear Model (P = 0.26):

$$\text{Yearly incidence (sarcomas/person·year)} = 2.0 \times 10^{-5} \underline{D} \qquad (6)$$

Dose-Squared x Exponential Model ($P = 0.27$)

Yearly incidence (sarcomas/person·year) $= 18 \times 10^{-8} D^2 e^{-0.0015 \underline{D}}$ (7)

where for bone sarcomas, D is the intake to blood in uCi ^{226}Ra plus 2.5 times the intake in uCi ^{228}Ra.

The linear Equation 6 from Subgroup B predicts a total of 1.25 bone sarcomas (1.06 induced plus 0.19 natural) in the Subgroup B female dial workers with intakes to blood below 50 microcuries. However, applying this equation to the 1,321 Group A female dial workers, with intakes below 50 microcuries and much longer assigned years at risk, predicts a total of 4.45 bone sarcomas (4.07 induced plus 0.38 natural). For an expectation of 4.45 sarcomas, there is only a 1.2% chance that zero sarcomas will occur ($P = 0.012$). Hence, we regard linear Equation 6, 2×10^{-5} D, as a reasonable upper limit to the risk from radiation induced bone sarcomas at low intakes.

We regard the actual risk from bone sarcoma at low intakes of radium to be somewhere between our upper limit (linear Equation 6) and the lower limit of zero. We tentatively assume it to be midway between our upper and lower limits.

The provisional best estimate of bone sarcoma risk from ^{226}Ra is:

Yearly incidence (bone sarcomas/person·year) $= 1 \times 10^{-5} D$ (8)

The provisional best estimate predicts 2.41 total expected bone sarcomas (2.03 induced plus 0.38 natural) among the 1,321 female dial painters of Group A with intakes below 50 mcirocuries. For an expectation of 2.41 sarcomas, there is a 9% chance that zero sarcomas will occur. Hence, Equation 8 cannot be statistically rejected ($P > 0.05$).

It is possible that one or more new bone sarcomas may occur in the low-intake persons under present surveillance. In fact, one or more sarcomas at low intake may already exist among the 24 women with known cases of bone sarcoma who were buried without an evaluation of radium content (Gustafson and Stehney 1983; see p. 162). Therefore, it is important to exhume and measure as many of the 24 buried but unmeasured bone sarcoma patients as possible and to continue to follow the living exposed persons. If no additional sarcomas are found, it would suggest that our provisional best estimate of bone sarcoma risk may be too high, whereas finding more sarcomas may suggest that our tentative best estimate is about right or perhaps too low. The present study is unique in that it offers the only known opportunity to resolve this question of scientific and humane importance.

2. Cumulative Risk from Lifelong Intake of Radium

The following cumulative risk estimates for long-lived ^{226}Ra and ^{228}Ra correspond to a constant concentration of radium in bone throughout life -- at the equilibrium concentration resulting from the adult intake to blood of 1 pCi of a radium isotope per day. The daily intake required to maintain a constant concentration of radium in bone may differ for infants, children, adolescents and adults. The general finding that the $^{226}Ra/Ca$ ratio in bone remains roughly constant with age (Fisenne and Keller 1979, Rajewsky et al. 1965) probably reflects a more-or-less constant ratio of $^{226}Ra/Ca$ in the diet at environmental levels during much of the lifespan (Wrenn 1977, Wrenn et al. 1984). Even the recent results of Muth and Globel (1983), which suggest possible peaks in ^{226}Ra concentration at about one and ten years of age, correspond to an average lifespan concentration that is only about 20% higher than the average of their adult values. For simplicity, we assume that the daily intake of dietary ^{226}Ra and ^{228}Ra at environmental levels results in constant Ra/Ca ratios in bone. For long-lived ^{226}Ra ($T_{1/2}$ = 1,600 years) and ^{228}Ra ($T_{1/2}$ = 5.77 years), one might expect the high rate of Ra accretion in the growing bones of a child to be opposed by the enhanced rate of Ra removal from resorbing bone.

However, short-lived ^{224}Ra ($T_{1/2}$ = 3.62 days) should decay before appreciable resorption occurs. Therefore, we assume that skeletal concentrations of ^{224}Ra in children (aged 0 to 15) and in adolescents (aged 16 to 20), would be three and two times, respectively, the adult concentrations, based on the fractions of injected ^{224}Ra estimated to decay in bone (Spiess and Mays 1970).

In the following pages, the risks from the lifelong intakes to blood of 1 pCi Ra/day will be estimated for ^{226}Ra (head carcinomas and bone sarcomas), ^{228}Ra (bone sarcomas only) and ^{224}Ra (bone sarcomas only).

^{226}Ra - Cumulative Risk from Head Carcinoma:

For a lifetime of 75 years, only the ^{226}Ra intake during the first 65 years is considered effective for the induction of observable head carcinomas because of the assumed minimal latent period of 10 years for these carcinomas (Rowland et al. 1978).

$$\begin{array}{l} ^{226}\text{Ra} \\ \text{cumulative} \\ \text{intake to} \\ \text{blood} \end{array} = \left[\frac{10^{-6}\ \text{microCi}}{\text{day}}\right]\left[\frac{365\ \text{days}}{\text{year}}\right]\left[65\ \text{years}\right] \qquad (9)$$

$$= 0.0237\ \text{microcuries}$$

The risk rate for observable head carcinomas builds up linearly, from zero at age 10 (the minimal latent period) to a maximum 65 years later at the end of life. From Equations 4 and 9:

$$\begin{array}{l} \text{Risk rate at} \\ \text{end of life} \end{array} = \left[\frac{1.6 \times 10^{-5}}{\text{microCi year}}\right]\left[0.0237\ \text{microCi}\right] = \frac{0.380 \times 10^{-6}}{\text{year}} \qquad (10)$$

$$\begin{array}{l} \text{Av. risk rate} \\ \text{during expression} \\ \text{period} \end{array} = \frac{0 + 0.380 \times 10^{-6}/\text{year}}{2} = \frac{0.190 \times 10^{-6}}{\text{year}} \qquad (11)$$

$$\text{Cumulative risk } (^{226}\text{Ra head carc.}) = \left[\frac{0.190 \times 10^{-6}}{\text{year}}\right]\left[65 \text{ year}\right] = 12 \times 10^{-6} \qquad (12)$$

In words, if one million persons each had the equivalent intake throughout life of 1 pCi ^{226}Ra/day to blood, about 12 cases of induced head carcinoma would be predicted. For a natural incidence rate, averaging 0.5×10^{-5}/year, about 375 naturally occurring head carcinomas should occur among one million persons during an average lifespan of about 75 years.

^{226}Ra - Cumulative Risk from Bone Sarcoma:

For a lifetime of 75 years, only the ^{226}Ra intake during the first 70 years is considered effective for the induction of observable bone sarcomas because of the assumed minimal latent period of 5 years for bone sarcomas (Rowland et al. 1978).

$$^{226}\text{Ra cumulative intake to blood} = \left[\frac{10^{-6} \text{ microCi}}{\text{day}}\right]\left[\frac{365 \text{ days}}{\text{year}}\right]\left[70 \text{ years}\right] \qquad (13)$$

$$= 0.0256 \text{ microcuries}$$

The risk rate for induced bone sarcomas builds up linearly, from zero at age 5 (the minimal latent period) to a maximum 70 years later at the end of life. From Equations 8 and 13:

$$\text{Risk rate at end of life} = \left[\frac{1 \times 10^{-5}}{\text{microCi year}}\right]\left[0.0256 \text{ microCi}\right] \qquad (14)$$

$$= 0.256 \times 10^{-6}/\text{year}$$

$$\begin{array}{l}\text{Av. risk} \\ \text{rate during} \\ \text{expression} \\ \text{period}\end{array} = \frac{0 + 0.256 \times 10^{-6}/\text{year}}{2} = \frac{0.128 \times 10^{-6}}{\text{year}} \tag{15}$$

$$\begin{array}{l}\text{Cumulative} \\ \text{risk (}^{226}\text{Ra} \\ \text{bone sar.)}\end{array} = \left[\frac{0.128 \times 10^{-6}}{\text{year}}\right] \left[70 \text{ years}\right] = 9 \times 10^{-6} \tag{16}$$

In words, among one million persons so exposed, about nine cases of induced bone sarcoma would be predicted. For a natural incidence rate, averaging 1 x 10^{-5}/year, about 750 naturally occurring bone sarcomas should occur among one million persons during an average lifespan of 75 years.

A total of 21 ^{226}Ra-induced cancers (12 carcinomas plus 9 sarcomas) is predicted for this exposed population.

^{228}Ra - Cumulative Risk from Bone Sarcoma:

Following the above procedures, but assuming that each microcurie of ^{228}Ra is equivalent to 2.5 microcuries of ^{226}Ra for bone sarcoma induction (Rowland et al. 1978), about 2.5 x 9 or 22.5 bone sarcomas should accumulate during the lifespan of one million persons, each with an intake to blood of 1 pCi ^{228}Ra/day.

^{224}Ra - Cumulative Risk from Bone Sarcoma:

The intravenous injection of 1 microcurie ^{224}Ra gives an alpha particle dose of about 0.2 rad averaged over the 7-kg marrow-free skeleton of adult reference man (Spiess and Mays 1970). Thus, the adult intake to blood of 1 pCi ^{224}Ra/day, or 365 x 10^{-6} microcuries/year, gives an average skeletal dose rate of 73 x 10^{-6} rad/year to adult man. However, based on the fractions of intravenously injected ^{224}Ra which are estimated to decay in juvenile, adolescent and adult bone (Spiess and Mays 1970), we assume that the skeletal concentrations of short-lived ^{224}Ra are three times higher in children under age 16, and two times higher in adolescents aged 16 to 20, than in adults. The dose within each age interval is:

Age	Years in interval	Dose rate (rads/year)	Rads in interval
Under 16	16	3 x 73 x 10^{-6}	0.00350
16 to 20	5	2 x 73 x 10^{-6}	0.00073
21 to 70	49	1 x 73 x 10^{-6}	0.00358
Cumulative	70		0.00781

Using the cumulative risk coefficient for ^{224}Ra of 200 bone sarcomas/10^6 person·rad of average skeletal dose, which life table analysis has shown to be fairly constant for injection ages ranging from 1 to 70 years (Mays 1983):

$$\begin{array}{l}\text{Cumulative}\\ \text{risk } (^{224}\text{Ra}\\ \text{bone sar.})\end{array} = \left[\frac{200 \text{ bone sar.}}{10^6 \text{ person}\cdot\text{rad}}\right]\left[0.00781 \text{ rad}\right] = 1.6 \times 10^{-6} \qquad (17)$$

Among one million persons so exposed, about 1.6 induced bone sarcomas should be expected. Alternatively, only 1.0 induced sarcoma would be expected if the concentration of ^{224}Ra in bone remained constant at the adult equilibrium concentration.

For a 1-pCi/day input to blood for one million people, the predicted 1.6 bone sarcomas for ^{224}Ra are much lower than the predicted 9 bone sarcomas for ^{226}Ra. This is primarily because the short 3.62-day half-life of ^{224}Ra limits the amount of radiation dose it can deliver. However, the short half-life of ^{224}Ra causes much of it to decay on bone surfaces where it has an enhanced effectiveness per rad of average skeletal dose.

Relative Toxicities and Occurrence of ^{228}Ra and ^{226}Ra:

In the interim regulations for radioactivity in drinking water, ^{228}Ra is coupled to ^{226}Ra. As shown in Figure II-3, analysis for ^{228}Ra is not made unless the ^{226}Ra concentration exceeds 3 pCi/L. This procedure is used because the extra cost of analyzing for both ^{228}Ra and ^{226}Ra was considered excessive. The other two factors that bear on this situation are the relative toxicities of the two isotopes and the occurrence of ^{228}Ra.

The difference between the relative risk of ^{226}Ra and ^{228}Ra can be inferred from bone sarcoma data for beagles (Mays 1980). A linear fit to the data indicates a ^{228}Ra risk of 900 bone sarcomas/10^6 beagle-rad and a ^{226}Ra risk of 320 bone sarcomas/10^6 beagle-rad. This would suggest that ^{228}Ra is about 2.8 times more toxic than ^{226}Ra. A more recent estimate from the same group of beagles puts the factor at 2.0 ± 0.5 (Wrenn et al. 1984). The modified ICRP dosimetric model suggests that ^{226}Ra is more toxic than ^{228}Ra (see Table VIII-4).

When the interim regulations were promulgated, it was assumed that the toxicities of ^{226}Ra and ^{228}Ra were approximately equal. However, as discussed in Section V-C, the Argonne model (Marshall and Groer 1977) predicted that ^{228}Ra would be more radiotoxic than ^{226}Ra. A number which is sometimes used is that ^{228}Ra is 2.5 times more toxic than ^{226}Ra (Rowland et al. 1978).

The Rowland et al (1978) comparisons refer only to bone sarcomas, not total toxicity, and only to cases of short periods of intake followed by accumulation of radiation dose during remaining life. Relative to ^{226}Ra the radiation dose from ^{228}Ra accumulates more quickly and ^{228}Ra delivers a greater dose to bone surface per microcurie of intake, although the mean skeletal doses become equal at about 50 years after intake. Therefore, per microcurie or per rad of mean skeletal dose, greater effectiveness for bone sarcomas would be expected for ^{228}Ra after a limited period of intake; Rowland et al (1978) estimated 2.5 on a microcurie basis and 1.5 on a rad basis.

Radium is also kown to have caused head carcinomas in dial painters. However, head cancers are associated with ^{226}Ra and not with ^{228}Ra. This increases the toxicity per microcurie from ^{226}Ra relative to that from ^{228}Ra.

It has been shown that the ^{226}Ra/^{228}Ra ratio in aquifers is not constant and may vary by a factor of 14 within an area of less than 5 square miles (Michel and Moore 1981). Little seasonal variation was seen, and within a 4-km^{2} area, ^{228}Ra ranged from 4.71 to 11.9 pCi/L, while ^{226}Ra ranged from 2.71 to 26.53 pCi/L. The ^{226}Ra/^{228}Ra ratios varied between 0.23 and 3.0, with most being between 1.2 and 1.8.

Recent data from Iowa indicate somewhat similar values: ^{228}Ra ranged from 0.6 to 5.3 pCi/L, ^{226}Ra ranged from 0.3 to 24.6 pCi/L, and the ^{228}Ra/^{226}Ra ratio varied between 0.26 and 7.6.

Although there is little information describing the occurrence of ^{228}Ra in water supplies, a recent study (King et al. 1982) has been made of 150 water supplies in South Carolina. This study concludes that roughly 40% to 50% of the groundwater wells with total radium in excess of 5 pCi/L in South Carolina may be overlooked due to the present coupling of ^{226}Ra and ^{228}Ra.

In order to determine whether ^{226}Ra and ^{228}Ra should be considered separately in the revised regulations, the EPA is currently conducting a study of the occurrence of ^{228}Ra.

This survey is being done primarily in the areas of high thorium content. Since thorium is a 100% isotope ^{232}Th, which decays to ^{228}Ra, and since thorium is highly insoluble, these are the most likely areas for ^{228}Ra to be found in water.

C. Does a Threshold in the Dose-Response Curve Exist?

Although there appear to be thresholds for acute effects from toxic substances, it may be that there is no similar threshold for cancer. After examining several types of cancer, Schneiderman et al. (1979) concluded that they had found "little or no evidence of thresholds in human cancer." However, a "practical" threshold for cancer due to ingestion has been suggested in the study of radium watch dial painters (Evans 1974).

Schneiderman et al. (1979) argue that these "practical" thresholds are defective, or at least unrepresentative, in at least two ways. First, most data involve high incidences, while the lifetime probability of cancer in man is low. The authors question whether the data from these high-incidence situations can really be applied to the possible effects of low-level radiation in producing cancer. They also point out that for high doses (such as those received by radium watch dial painters), the radiation may kill the host before other tumors can become clinically noticeable. Thus, the effect could be just an artifact of high doses.

The contention that a threshold exists for the radium watch dial painters (Evans 1974) comes from noting that no cases were found below average doses of 1,000 rads. However, the doses are not inconsistent with a linear dose-response curve going through the origin. In addition, the radium dial painter data may be biased, since there are 30 known cases of persons with tumors who died without radioassay. The possibility that some of these cases might be found to fall in the low-dose region cannot be ruled out.

In an attempt to better understand the data for radium watch dial painters and beagles which have ingested radium, a third axis has been added to the dose-response curve (Raabe et al. 1980). This new axis, the time to death, reveals what is often referred to as a "practical" threshold. At lower dose rates, the time to death increases; conversely, as the dose rate increases, the lifetime decreases. When the time to death equals the average lifetime, a "practical" threshold is said to exist. The thresholds determined by this approach are 28 rem/year for man, 80 rem/year for beagles and 110 rem/year for mice. The data used have the same uncertainty at low doses, but this is only a new approach for interpreting old data. The old question regarding the shape of the curve is not answered by this new analysis. The shape of the curve at low doses is not clear; it is possible that it is not a straight line and that there is no "practical threshold." Although this new analysis is interesting, it does not appear to add any new information concerning health effects at low doses.

A statistical analysis result that is often called a practical threshold is related to the standard deviation in cancer rate. Any numerical value has an uncertainty. For cancer rates, this uncertainty is due to arbitrary reporting times (those reported on December 31 are listed in one year, while those on January 1 are reported for the next year), variations in reporting procedure, information that is not reported and other causes. Thus, the actual number of cancers from all causes is a number with some uncertainty. When the estimated number of excess cancers due to a cause such as radium in drinking water is less than the uncertainty in the overall number, one could sugggest that a "practical threshold" has been reached. The use of the term "threshold" in the sense described here seems inappropriate and can lead to misleading conclusions. It would seem better to use the phrase "acceptable variation."

It appears that the existing information is not good enough to determine whether or not a threshold exists. Furthermore, it is possible that there will never be enough information to determine the possible existence of a threshold

(Taylor 1980) because of the large number of animals or humans required. The existing data can only suggest possibilities.

Observing the lack of convincing evidence to the contrary, the EPA supports the no-threshold position (see Appendix). The courts have recently upheld the principle that there is no threshold exposure level for a carcinogen (Reserve Mining Co. versus EPA, 514 F.2d at 508, notes 24 and 25; EDF versus EPA, 598 F.2d at 88). This position is also supported by the Interagency Regulatory Liasion Group (IRLG) on risk assessment, which argues that if a threshold did exist, the wide variation of individual susceptibility would make it extremely difficult to determine one acceptable level.

D. The Uncertainty of the Risk Estimate

Although error bars are included for some of the data discussed here, the level of confidence given to these values is not clear, and all possible contributions to the error or uncertainty have not been included. In general, possible confounding factors and bias have not been examined. The EPA has estimated that the calculations relating to ingestion rate and risk in the interim regulations have an uncertainty of at least a factor of four(USEPA 1984).

Using the difference in risk determined by the absolute and relative risk models, BEIR III suggests that the uncertainty is in the range of a factor of three (NAS 1980). A number of factors contribute to the uncertainty in the risk estimate for ingestion of radium. Besides the uncertainty involved in the uptake, retention, excretion and location of radium in the bone structure, there are also uncertainties due to physiologic and metabolic changes with age, differences in nutrition, calcium and phosphorous levels and vitamin balance and the clinical and experimental differences between researchers.

It is often suggested that an understanding of the magnitude of the effects of low-level radiation can be derived from epidemiology studies. However, the necessary sample sizes are impractically large. Giving some idea of the difficulties involved, Land (1980) states that "if the excess risk is proportional to dose, and if a sample of 1,000 persons is necessary to determine the effect of a 100 rad exposure, a sample of 100,000 may be needed for a 10 rad exposure and about 10 million for 1 rad." Using the relationship on which the above estimates were made, a sample of 10 million persons would have to be analyzed for 530 years if the exposure were due to 5 pCi/L of radium in drinking water.

An uncertainty of a factor of four to five appears to be a reasonable estimate from background studies dona as a part of the development of the radiation regulations under the Clean Air Act(USEPA 1984).

E. Acute Health Effects[a]

There are three general areas in which radioactivity produces deleterious effects on humans. Examples of the effects in these areas include:

- Developmental and Teratogenic Effects
  - Effects on the fetus
  - Developmental abnormalities (skeleton and central nervous system)
  - Embryo lethality
- Genetic Effects (effects in subsequent generations)
  - Mutagenic changes in DNA
  - Hereditary effects
  - Diseases caused by mutations
    - Dominant/recessive diseases
    - Chromosomal anomalies

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[a] For the health effects of ionizing radiation, see:

Known Effects of Low-Level Radiation Exposure. April, 1980. U.S. Department of Health, Education and Welfare, Public Health Service, National Institutes of Health. NIH Publication No. 80-2087.

The Effects on Populations of Exposure to Low Levels of Ionizing Radiation. 1972. Report of the Advisory Committee on Biological Effects of Ionizing Radiations (BEIR), National Academy of Sciences, Washington, D.C. 20006.

Drinking Water and Health, Safe Drinking Water Committee. 1977. National Academy of Sciences, Washington, D.C. 20006.

Interagency Task Force on Ionizing Radiation. February 27, 1979. U.S. Department of Health, Education and Welfare (Labassi Report).

Casarett AP. 1968. Radiation Biology. Prentice-Hall, Inc., Englewood Cliffs, New Jersey.

- Somatic Effects (effects in the person exposed)

  Carcinogenesis (including leukemia)
  Cataract of the lens of the eye
  Non-malignant damage to the skin
  Gonadal cell damage/impairment of fertility
  Lifespan shortening

The acute effects of ionizing radiation appear within 30 days as a variety of tissue changes or syndromes. Some effects are lethal, and some are not.

Doses to animals of 100,000 rad or more usually cause inactivation of many substances that are needed for the basic metabolic processes of the cell and tissues, and thus lead to an immediate death. Doses of about 10,000 rad produce hyperexcitability, incoordination, respiratory disease and possible damage to the nervous system, and lead to death in a day or two. In the dose range from 900 to 10,000 rad, most animals die in three to five days due to morphologic changes or damage to the gastrointestinal (GI) tract. For doses in the range of 300 to 900 rad, death usually occurs in 10 to 15 days due to alterations in blood cells and the blood-forming organs (hematopoietic system).

Exposure of humans to doses of 50 rad or greater leads to radiation sickness. This is characterized by headache, dizziness, malaise, abnormal sensations of taste or smell, nausea, vomiting, diarrhea, decreased blood pressure, decrease in white blood cells and blood platelets, increased irritability and insomnia. Exposures of several thousand rads or more cause shock, abdominal cramps, cyanosis, coma and death. For doses in the range of 500 to 2,000 rad, normal food and fluid intake is depressed, followed by dehydration, hemoconcentration, circulatory collapse and death. Nausea, vomiting and some diarrhea can result from exposures of less than 500 rad. The body then seems to recover, but a few weeks later, chills, fatigue, hemorrhages in the skin, ulceration of the mouth, pharynx and intestine, impairment of immune mechanisms and hemorrhagic ulceration set in, permitting entry of bacteria. Death, if it occurs, is usually between the third and sixth week.

Embryos have been shown to be especially susceptible to ionizing radiation. Those exposed *in utero* at Nagasaki and Hiroshima showed microcephaly (small brain) and mental retardation. Other exposed children suffered from congenital dislocation of hips, Mongolism and congenital heart disease.

The nervous system of humans is usually found to be extremely radioresistant in terms of morphologic changes, but it does demonstrate a variety of physiologic responses to relatively low doses of radiation. These effects include changed reception activity of the eye and changes in conditional reflex activity.

To understand genetic effects, let us examine how life begins. The creation of new life is determined by the union of a sperm cell and an egg cell to form a single fertilized egg. This is followed by millions of cell divisons. The blueprint for these cell divisions and for how the new life grows and develops is found in the DNA molecule. The DNA molecule is a double helix string of atoms and carries the basic genetic information. The basic units of the DNA molecule are four bases, Guaninc (G), Cytosine (C), Adenine (A) and Thymine (T). Ionizing radiations can change the structure of the DNA molecule by changing the way the atoms are bonded or connected together (this occurs through the atomic electrons). For example, two bases (such as T) could be in adjacent steps instead of alternate steps. In that case, ionizing radiation could break an electronic bond, and the T bases would bond to each other instead of the structure as shown. Following that change, the cell divisions would follow a new genetic blueprint determined by the new DNA structure. This new pattern almost always leads to deleterious effects.

Nuclear radiation's ability to ionize atoms is important for human health effects. The direct effect is thought to be the ionization of the atoms of DNA in the cell, which changes the cell's behavior. An indirect effect may occur when water in the cell is ionized. Human cells are about 75% water, and when water is ionized, it can produce the highly reactive HO ion (called the hydroxyl free radical). The HO can attack and damage the DNA.

Agents that can change the genetic code (called mutagens) can be found inside our bodies (these mechanisms are not well understood) or outside our bodies. External mutagenic agents include chemicals, drugs, elevated temperatures and ionizing radiation.

Genetic effects of ionizing radiation include abnormalities, recessive diseases (in which both chromosomes in the pair have some defect) and chromosome damage. Some examples of these maladies are listed below:

- Abnormalities
  - Extra fingers and toes
  - Short-lived dwarfism
  - Progressive involuntary movements
  - Mental deterioration
  - Several kinds of anemia
- Recessive Diseases
  - PKU -- a form of mental deficiency
  - Tay-Sach's disease (leads to blindness and death in the first few years of life)
  - Sickle cell anemia
  - Cystic fibrosis
  - Recessive mutations located in the X chromosome (noted almost exclusively in males, since males only have one X chromosome)
    - Hemophilia
    - Color blindness
    - A severe form of muscular dystrophy
- Chromosome damage
  - Too many or too few chromosomes can lead to embryonic death or miscarriage
  - Broken chromosomes can be involved in:
    - Diabetes
    - Schizophrenia

In general, it is believed that mutations, whether spontaneous or induced, can be harmful even though the harm may be trivial and the effect may not show up for hundreds of generations. Each new harmful mutation is eventually eliminated by reduced viability or gene extinction.

The number of mutations appears to be proportional to the dose, and since spontaneous mutations do occur, the concept of doubling dose is used; specifically, the doubling dose is that dose of ionizing radiation that will produce mutations equal in number to those occurring spontaneously. The doubling dose for genetic effects of ionizing radiation is in the range of 20 to 200 rem. In the U.S., a cumulative dose of 5 rem/generation (or 170 mrem/year for 30 years - the child-bearing range) might produce 60 to 1,000 genetically determined illnesses of various sorts per million live births. About 4% of live-born infants (or 40,000) show evidence of hereditary defects. Thus, if this estimate is reliable, ionizing radiation can cause a 0.1% to 1.6% increase over the expected incidence of genetically determined illnesses. If the same exposure level is continued for several generations, the percentage of excess illnesses will increase due to the presence of the malady in the parents, thus increasing the probability of it occurring in the offspring. The effect level eventually might reach 300 to 7,500 cases per million live births, a 0.5% to 12.5% increase.

Of the somatic effects due to ionizing radiation, the most important are cancers. The mechanism causing cancer is not known at this time. Thus, statistical data from animal and human epidemiological studies must be used. Again, little is known about the actual effects of low doses; however, by extrapolating from high doses, the death rates can be predicted, as shown in Table VI-3.

The estimates listed in Table VI-3 do not include several variables which will change the predicted effect level. The effect on the fetus or child is known to be higher (perhaps by a factor of three to five). The effects shown have different latency periods varying from 5 to 25 years.

Other factors that can modify the predicted somatic effects of low-level radiation are dose rate and biological variation. In general, the effect of ionizing radiation on the human body is cumulative. This would imply that receiving 100 rem in a day would have the same effect as receiving 1 rem/day for 100 days. This is not true at high rates, where receiving 50 rem/minute for one minute is three times more destructive than receiving 1 rem/minute for 50 minutes. Biological variation is due to the different sensitivities of body tissues and the existence of some repair mechanisms in the body.

Table VI-3 Expected Death Rates for Somatic Diseases

Disease	Number of deaths/10^6 people exposed/year/rem
Cancers	
Lung	0.4-1.5
Breast	1.5-6.0
Skeleton	0.1-0.3
GI and Stomach	0.5-1.0
Overall	2.5
Leukemia	2-4
Leukemia (*in utero* exposure)	25

F. Discussion

As the preceding analysis has shown, a considerable amount of conflicting and contradictory evidence exists concerning the health effects of ingested radium. In light of the uncertainties in the data and models, the situation is not entirely clear. Even for the single case of radium ingestion by humans, no functional relation uniquely fits the dose-response curve. Although a dose-squared relation gives an excellent fit, a linear component in the low dose region cannot be ruled out.

In developing the interim regulations, the EPA used the linear extrapolation of the dose-response curve to represent the likely upper limit of the possible effect levels(see Appendix for the policy statement used). This should not be taken to imply that EPA believed (or believes) that this was either the most correct or the best possible fit. Rather, the linear fit was chosen as the one most appropriate for standard setting.

The linear non-threshold model was brought about by the necessity of making quantitative estimates of the effect of fallout. It is a simple approach, and it appeared to be the most prudent choice. The caveat that the reality may be that the risk is higher than that predicted by the linear dose-response curve assumption must not be forgotten.

Recognizing that the only exposure guaranteeing maximum protection of human health is zero, EPA does not have an "acceptable" risk level. The Office of Drinking Water, on the other hand, considers both non-carcinogenic and carcinogenic health effects, and the excess carcinogenic risk range under consideration is 10^{-4} to 10^{-6}/lifetime.

In its recent decision involving benzene, the U.S. Supreme Court (July 2, 1980) stated that "if the odds are one in a thousand...a reasonable person might well consider the risk significant and take appropriate steps to decrease or eliminate it." In the same decision, they also stated that "if, for example, the odds are one in a billion that a person will die from cancer by taking a drink of chlorinated water, the risk clearly could not be considered significant." These observations have been taken from dicta, and thus are out of context, but they do give an idea of the possible bounds.

There exists no policy at EPA concerning the acceptable risk level or a general target level. Using the Safe Drinking Water Act, the standards (MCLs) are to be set using health effects, while also taking costs and feasibility into account. Few other standards allow a risk as high as 10^{-4} fatalities/lifetime, as does the MCL for radium in drinking water. However, the MCL for trihalomethanes in drinking water and the vinyl chloride air standard both allow risks of the order of 10^{-4} fatalities/lifetime. The MCL for radium in drinking water was set on the basis of health effects and costs. In view of the trend in risk analysis of examining concentrations of radioactivity that lead to risks in the range of 10^{-5} to 10^{-7} fatalities/lifetime, it is difficult to argue that the MCL for radium that leads to a risk of 4.4×10^{-5} fatalities/lifetime is over-protective.

Data from studies involving animals such as mice and dogs and human data were used to determine the MCL for radium. That the effects of a chemical in an animal are applicable to man has been recommended by the National Academy of Sciences and upheld in recent court decisions (Reserve Mining Co. versus EPA, 514 F.2d at 514; EDF versus EPA, 598 F.2d at 87; Ethyl Corp. versus EPA, 541 F.2d at 26).

According to ICRP 23 (1975), most of the radium is ingested in the form of food (90% in normal man). Under normal conditions with only about 10% of the radium in humans is ingested from drinking water with concentrations of the order of a few pCi/L or less.

It is known that the dose relationships that hold true for animals do not necessarily apply, in the scientific sense, directly to humans. Some of the problems encountered in using animal data to predict effects in humans include (Schneiderman et al. 1979):

- Animal populations are often inbred, whereas human populations are hetrogenous.
- Relative size -- volume and surface area.
- Different plasma protein and tissue binding of elements and compounds.
- Different generation time of cells and repair rates of test animals living in a controlled environment.
- The lifespans of animals are different than those of human beings.

In setting the MCL for radium,the data from human exposure were used with animal data support.

One approach to setting standards for protecting health is to use the occupational standard and divide by 3, 10 or 100 (Rowland et al. 1979). This approach is rejected by the EPA Office of Drinking Water because of its arbitrary character and because the occupational exposure calculations do not include age effects (increased susceptibility of fetuses and children to exposure before achieving a working age). This approach was used by the Canadians (Guidelines 1978) in setting the standard at 1% of the ICRP value. This yielded a value of 27 pCi/L as the maximum acceptable concentration. The Canadian objective concentration is 1/10 of this value, or 3 pCi/L. No variance and exemption levels such as the EPA's MCL, exist for the Canadian maximum acceptable concentration.

After all the considerations described above, it is believed that the following values are the best available for standard setting purposes: 5 pCi/L consumed at 2 L/day, produces a body burden of 250 picocuries and a bone dose of 92 millirem/ year, leading to a risk of 4.4 excess bone and other cancer deaths/10^5 people exposed/lifetime. These values were determined using a modified ICRP 30 model (Sullivan et al. 1981).

G. Conclusion

The present MCL for the combined concentrations of ^{226}Ra and ^{228}Ra is 5 pCi/L. This level was set on the basis of health effects and costs (USEPA 1976). As was pointed out, we are considering separating the analysis required for ^{226}Ra and ^{228}Ra (see Figure II-3). After examining the analyses and data that have emerged since the interim regulations for radioactivity in drinking water were promulgated, it is concluded that at this time, one cannot make a clear choice between the possible alternative dose-response curves, nor can one decide whether a threshold exists. These choices must be made by policy decisions and may well be influenced by such aspects of regulatory decision making as economics, politics, feasibility of treatment and social impact.

VII. MECHANISMS OF TOXICITY

Knowledge about the basic methods of interaction of radiation with matter at both the cellular and the molecular level has recently increased: It may now be sufficient to allow us to localize the targets for the initiation of cancer at the level of individual particle tracks, ionizations and particular bonds in the DNA molecule.

It has been suggested that the cancer causing mechanism involves two initiation events in one flattened endosteal cell at or near the bone surface. A promotion event, cell killing, and cell replacement are involved. These mechanisms provide an excellent fit over the whole dose-time-response surface to data for induction of osteosarcoma in dog and in man by alpha particles of radium, including ^{226}Ra, ^{228}Ra and ^{224}Ra (Marshall and Groer 1977, 1978). The Marshall-Groer theory suggests that the dose range between roughly 20 to 40 rads and at 140 rads is the region where the alpha particles seem to yield a dose-squared relationship. This supports the finding of Rowland et al. (1978) that the human osteosarcomas caused by long-lived radiation were well-fit by the squared-exponential function of injured activity.

The question to be determined in the theory of the mechanism of bone cancer is the relationship between the radiation and the cross-section of the target. The question is, what is the target - is it a cell, DNA or a cell nucleus? It appears that at doses greater than about 40 rads, two DNA targets are more likely to be hit by two independent alpha particles than by one. This leads to a dose-squared response. However, for doses of less than 40 rads, it appears to be more likely that both DNA molecule targets will be hit by one alpha particle, leading to a linear dose-response. If the Marshall-Groer theory that there are two targets and that the whole nucleus is the volume within which they are randomly contained is correct, then low LET radiation would show a squared response down to as low as one rad and would be about two orders of magnitude less dangerous at environmental levels than current estimations on the basis of the linear model. However, one rad of alpha radiation corresponds to about 20 rems. Thus, it is expected that at the level for the existing MCL for radium, the most likely model could well be a linear one.

When small amounts of radium are ingested in human beings, they are deposited primarily in skeletal tissue. For such ingested radium, particularly ^{226}Ra, there are two potential types of malignancies: (1) bone sarcomas which primarily consist of osteosarcomas, fibrosarcomas and chondrosarcomas and (2) head carcinomas, which are primarily carcinomas of the paranasal sinuses or of the mastoid air cells.

It appears that when humans ingest ^{224}Ra and ^{228}Ra, no head carcinomas occur (Rowland 1978). The suggestion here is that ^{226}Ra decays to radon-222 gas within the body, and that the gas then goes to the head cavity. The suggestion is that the gas, radon, is the major inducer of the carcinomas, because the pure ^{224}Ra and ^{228}Ra do not produce long lived radon gas.

Although it is clear that bone sarcomas are induced by ^{226}Ra ingestion, it appears that the incidence of leukemia induced is small. It has been noted that although it is possible that some leukemia has been induced by the ingested ^{226}Ra, the amount is smaller than had been previously expected. In a recent paper assembled at a National workshop, Mays (1983) deduced that the number of induced leukemias due to ingestion of ^{226}Ra is approximately a factor of ten lower than that predicted by the ICRP 30 report risk coefficient (ICRP 1977). The ICRP risk coefficient for alpha particles was approximately 400 leukemias per million person-rad. Mays (1983) further observed that there are several potential reasons why human leukemia induction is lower than had been previously thought:

1. Due to the non-uniform irradiation which allows undamaged stem cells to replace cells killed by irradiation.

2. Due to the low-frequency of transformable cells in the regions most heavily irradiated by alpha particles.

3. Becasue the RBE may be less than 20 for uniform alpha-emitting particles, as compared to sparsely ionizing radiation at low dose rates for leukemia induction.

4. Due to a lifespan risk coefficient of less than 20 leukemias per million person-rem for sparsely ionizing radiation at low doses and other potential factors.

Other potential carcinomas possibly resulting from the ingestion of radium have also been investigated. Multiple myeloma appears to be only slightly inducible by radium in human bones (Mays 1983). Other sites of potential carcinomas have been investigated, including breast, colon, lung, stomach, pancreas, cervix uterus, corpus uterus, rectum, red bone marrow and liver(Stebbings et al 1984). None of these cancers was observed to induce a statistically significant amount of carcinomas.

A. Mechanisms of Retention

In this section, we will discuss some of the mechanisms of uptake and retention of radionuclides in bone and their possible relevance to radiation toxicity. In general, there are two classes of bone seekers: those that seek bone surfaces and those that seek bone volumes. Plutonium, thorium, yttrium and americium are bone surface seekers, while the alkaline earths, calcium, strontium, barium and radium (226 and 228) are bone volume seekers. Because of its short half life, ^{224}Ra decays mostly on bone surfaces. Two mechanisms exist for the uptake of radium in the volume of the bone. The first is a surface exchange mechanism, and the second is a sort of self burial mechanism which leads to hot spots. It is known that the first uptake of calcium-45 following injection occurs at the bone surfaces in dogs and rabbits, but this calcium-45 rapidly returns to the blood (Marshall 1969). Bone formation occurs by apposition of new bone on the existing bone surfaces. Thus, if there is a concentration of radium in the blood and it is high, the bone which is forming will pick up large quantities of this activity, which are often called hot spots. In the first instance, this activity is located at or just under the growing bone surface. However, this radioactivity does not tend to stay in these bone surfaces, since it is usually buried by subsequently deposited bone. The new bone contains progressively smaller quantities of the activity because the specific activity in the blood would be continuously falling unless new radioactivity were ingested.

Because radium is deposited throughout the volume of bone, one would expect that the emitted radiations could reach the red bone marrow. It is this supposition that has led to estimates of leukemias due to ingested radium. As was pointed out earlier, there is some controversy about the contribution of radiation to red bone marrow and the possiblity of its leading to leukemias.

B. Thresholds

Although there appear to be thresholds for acute effects from toxic substances, it may be that there is no similar threshold for cancer. After examining several types of cancer, Schneiderman et al. (1979) found "little or no evidence of thresholds in human cancer." However, a "practical" threshold has been suggested for the cancers due to the ingestion of radium by watch dial painters (Evans 1974).

Schneiderman et al. (1979) argue that these "practical" thresholds are defective, or at least unrepresentative, in at least two ways. First, most of the data involve high incidences, while the lifetime probability of cancer in man is low. The authors question whether the data from these high-incidence situations can really be applied to the possible effects of low-level radiation in producing cancer. They also point out that for high doses (such as received by radium watch dial painters), the radiation may kill the host before other tumors can become clinically noticeable. Thus, the question remains unanswered. Thus, the effect could be just an artifact of high doses.

The contention that a threshold exists for the radium watch dial painters (Evans 1974) comes from noting that no cases count below-average doses of 1,000 rads. However, the doses are not inconsistent with a linear dose-response curve going through the origin. In addition, the radium dial painter data may be biased, since there are 30 known cases of persons with tumors who died without radioassay. The possibility that some of these cases might fall in the low-dose region cannot be ruled out.

In an attempt to better understand the data for radium watch dial painters and beagles which have ingested radium, a third axis has been added to the dose-response curve (Raabe et al. 1980). This new axis is the time to death. This analysis reveals what is often referred to as a "practical" threshold. At lower dose rates, it is observed that the time to death increases; conversely, as the dose rate increases, the lifetime decreases. When the time to death equals the average lifetime, a "practical" threshold is said to exist. The thresholds determined by this approach are 28 rem/year for man, 80 rem/year for beagles and 110 rem/year for mice. The data used have the same uncertainty at low doses, but this analysis is only a new approach for interpreting old data. The old question regarding the shape of the curve is not answered by this new analysis. The exponential function does appear to fit the data, but why should this be? The shape of the curve at low doses is not clear. It is possible that at lower doses, the curve is not a straight line and that there is no "practical threshold." Although this new analysis is interesting, it does not appear to add any new information concerning health effects at low doses.

A statistical result of analysis that is often called a "practical threshold" is related to the standard deviation in cancer rate. Any numerical value has an uncertainty. For cancer rates, these uncertainties are due to arbitrary time categories (those reported on December 31 are listed in one year, while those reported on January 1 go into the next year), variations in reporting procedures, information not reported and other causes. As a result, the actual number of cancers from all causes is a number with some uncertainty. When the estimated number of excess cancers due to a particular cause, such as radium in drinking water, is less than the uncertainty in the overall number, some suggest that a "practical threshold" has been reached. The use of the term "threshold" in the sense described here seems inappropriate and can lead to misleading conclusions. It would seem better to use the phrase "acceptable variation."

It appears that the existing information is not good enough to determine whether or not a threshold exists. Furthermore, it is possible that there will never be enough information to determine the possible existence of a threshold (Taylor

1980) because of the large number of animals or humans required. The existing data can only suggest possibilities.

Observing the lack of convincing evidence to the contrary, the EPA supports the no-threshold position. This policy is supported by recent court decisions. The courts have recently upheld the principle that there is no threshold exposure level for a carcinogen (Reserve Mining Co. versus EPA, 514 F.2d at 508, notes 24 and 25; EDF versus EPA, 598 F.2d at 88). This position is also supported by the IRLG Work Group on risk assessment. The argument is that if a threshold did exist, the wide variation of individual susceptibility would make it extremely difficult to determine one acceptable level.

VIII. QUANTIFICATION OF TOXICOLOGICAL EFFECTS

The quantification of toxicological effects of a chemical consists of an assessment of the non-carcinogenic and carcinogenic effects. In the quantification of non-carcinogenic effects, an Acceptable Daily Intake (ADI) is calculated. From this an Adjusted Acceptable Daily Intake (AADI) and Health Advisory (HA) values for the chemical are calculated to define the appropriate drinking water concentrations to limit human exposure. For ingestion data, this approach is illustrated as follows:

$$\text{ADI} = \frac{(\text{NOAEL or LOAEL in mg/kg/day})(\text{Body Weight in kg})}{\text{Uncertainty/Safety Factor}} = \text{mg/day}$$

$$\text{AADI} = \frac{\text{ADI}}{\text{Drinking Water Volume in L/day}} = \text{mg/L}$$

where:

NOAEL = no-observed-adverse-effect level.

LOAEL = lowest-observed-adverse-effect level.

Body weight = 70 kg for adult or 10 kg for child.

Drinking water volume = 2 L per day for adults or 1 L per day for children.

Uncertainty or Safety Factor = 10, 100 or 1,000

Utilizing these equations, the following drinking water concentrations are developed for non-carcinogenic effects:

1. A one-day HA for 10-kg child.
2. A one-day HA for 70-kg adult.
3. A ten-day HA for 10-kg child.
4. A ten-day HA for 70-kg adult.
5. A lifetime AADI for a 70-kg adult.

The distinctions made between the HA calculations (items 1 through 4) are associated with the duration of anticipated exposure. Items 1 and 2 assume a single acute exposure to the chemical. Items 3 and 4 assume a limited period of exposure (possibly 1 to 2 weeks). The HA values will not be used in establishing a drinking water standard for the chemical. Rather, they will be used as informal scientific guidance to municipalities and other organizations when emergency spills or contamination situations occur. The AADI value (item 5) is intended to provide the scientific basis for establishing a drinking water standard based upon non-carcinogenic effects.

A NOAEL or LOAEL is determined from animal toxicity data or human effects data. For animal data, this level is divided by an uncertainty factor because there is no universally acceptable quantitative method to extrapolate from animals to humans. The possibility must be considered that humans are more sensitive to the toxic effects of chemicals than are animals. For human data, an uncertainty factor is also used to account for the heterogeneity of the human population in which persons exhibit differing sensitivity to toxic chemicals.

An Office of Drinking Water (ODW) modification of the guidelines set forth by the National Academy of Sciences (NAS 1977, 1980b) is typically used in establishing uncertainty factors as follows:

- An uncertainty factor of 10 is used when good acute or chronic human exposure data are identifying a NOEL/NOAEL are available and are supported by acute or chronic toxicity data in other species.

- An uncertainty factor of 100 is used either when good acute or chronic toxicity data identifying a NOEL/NOAEL are available for one or more animal species (but human data are not available), or when good acute or chronic toxicity data identifying a LOAEL in humans are available.

- An uncertainty factor of 1,000 is used when limited or incomplete acute or chronic toxicity data are available, or when good acute or chronic toxicity data identifying a LOAEL (but not a NOEL/NOAEL) for one or more animal species.

The uncertainty factor used for a specific risk assessment is judgmental. Factors that cannot be incorporated in the NAS/ODW guidelines for selection of an uncertainty factor, but must be considered include: (1) the quality of the toxicology data,

(2) the significance of the adverse effect and (3) the existence of counterbalancing beneficial effects.

If toxicological evidence requires the chemical to be classified as a potential carcinogen (there is evidence to classify radium as a carcinogen following ingestion), mathematical models are used to calculate the estimated excess cancer risks associated with the ingestion of the chemical via drinking water. The bioassay data used in these estimates are from animal experiments. In order to predict the risk for humans from these data, it must be converted to an equivalent human dose. This conversion includes correction for non-continuous animal feeding, non-lifetime studies and for the difference in size. The factor that compensates for the size difference is the cube root of the ratio of the animal and human body weights. It is assumed that the average human body weight is 70 kg and that the average human consumes 2 liters of water per day. The multistage model is then fit to the equivalent human data to estimate the risk at low doses. The upper 95% confidence limit of this estimate is used. Excess cancer risks can also be estimated using other models such as the one-hit model, the Weibull model, the logit model and the probit model. There is no basis in the current understanding of the biological mechanisms involved in cancer to choose among these models. The estimates of low doses for these models can differ by several orders of magnitude.

The scientific data base used to calculate and support the setting of risk rate levels has an inherent uncertainty. This is because the tools of scientific measurement, by their very nature, involve both systematic and random error. In most cases, only studies using experimental animals have been performed. There is thus uncertainty when the data are extrapolated to humans. When developing risk rate levels, several other areas of uncertainty exist, such as (1) incomplete knowledge concerning the health effects of contaminants in drinking water, (2) the impact of test animal age, sex and species and the nature of target organ systems examined on the toxicity study results and (3) the actual rate of exposure of internal targets in test animals or humans. Dose-response data are usually only available for high levels of exposure, not for the lower levels of exposure for which a standard is being set. When there is exposure to more than one contaminant, additional uncertainty results from a lack of information about possible synergistic or antagonistic effects.

A. Non-Carcinogenic Effects

The knowledge base for non-carcinogenic effects is for low-LET radiation. There is an absence of information on non-carconogenic effects relating to high-LET radiation such as the alpha particle radiation from the naturally-occurring isotopes such as ^{226}Ra and ^{228}Ra. In any case, it would appear that the most important effects are the carcinogenic ones.

Besides cancer, the only other known health effects that may occur due to internally deposited radionuclides include non-stochastic effects, genetic effects and in utero developmental effects(UNSCEAR 1982).

In general, about 10 rads of low-LET radiation (or more) are needed to induce non-stochastic effects in humans(UNSCEAR 1982). This threshold is well above the known or anticipated levels or environmental exposure due to radium ingested from drinking water.

Using a conservative estimation approach, the low-LET doubling dose (dose needed to double the risk) for human genetic effects is 100 rad if the dose is delivered to the ovary or testis(USEPA 1984). Since little radium is deposited in the reproductive organs, the genetic effects would be expected to be negligible when compared to bone cancer.

The rate of teratogenic effects is in the range of 7.4×10^{-3} to 5.0×10^{-4} cases of mental retardation/live births/rad of low-LET radiation delivered between weeks 8 and 15 of gestation, with no threshold identified at this time (USEPA 1984). This effect is negligible in comparison to that due to bone cancer.

B. Quantification of Non-Carcinogenic Effects

In the absence or appropriate dose-responde data for non-carcinogenic effects, it is not possible to derive an AADI for ^{226}Ra and ^{228}Ra.

C. Carcinogenic Effects

Two isotopes of radium are of interest in the development of regulations relating to drinking water. These two isotopes are the alpha emitting ^{226}Ra and the beta emitting ^{228}Ra. These two isotopes are part of two independent radioactive series, the uranium and the thorium series.

In general, the effects of radiation are cumulative. For low LET radiation, this has been shown to be valid within a factor of about two or three. Radioisotopes such as radium are high-LET emitters because of the impact of the heavier particles on tissues.

As discussed earlier, radium is found primarily in groundwater supplies and in certain geological regions. A study is in progress to identify those community water supplies that would be likely to exceed a 5 pCi/L concentration of ^{228}Ra. Those that do exceed 5 pCi/L ^{226}Ra already have been identified. A preliminary report suggests that about 30% of the groundwater community water supplies have a reasonable likelihood of potential levels of ^{228}Ra that could exceed 5 pCi/L.

As detailed in ICRP 30 (ICRP 1979), radium that is ingested by humans is either excreted or deposited in the bone. Very little radium is deposited in other organs. The primary human health consequence of ingested radium is bone cancer. The effect on red bone marrow, in the form of leukemia, that is predicted by the dosimetric models for radiation in general appears to be about a factor of ten greater than that found in epidemiological studies. However, the risk rates predicted by dosimetric models and from epidemiological studies among radium watch dial painters are in good agreement. Studies in animals of bone cancer due to ingested radium also support the findings of the human epidemiology studies.

Human epidemiological data have been obtained for a group of women that ingested radium while painting radium on watch dials (Rowland et al, 1983). The incidence of bone sarcomas among 3055 female radium-dial workers who entered the dial industry before 1950 was used to determine dose-response relationships. Two subpopulations were analyzed: all measured cases who survived at least 5 years after the start of employment and all cases who survived at least 2-years after first incidence. The first a group was based on year of entry. It contained 1468 women who experienced 42 bone sarcomas; the expected number was 0.4. The second comprised a group based on time after first measurement. It contined 1257 women who experienced 13 bone sarcomas: the expected number was 0.2.

D. Quantification of Carcinogenic Effects

The risk and annual effective dose equivalent (EDE) for ingested ^{226}Ra and ^{228}Ra, using the modified ICRP 30 dosimetric model (ICRP 1977 and 1979 and Sullivan et al. 1981) and weighting factors derived from BEIR III (USEPA 1984), are tabulated in Tables VIII-1 and VIII-2.

For ^{226}Ra, as shown in Table VIII-1, the lifetime risk is 8.8 cases per pCi/L per million people exposed. The effective dose equivalent value of 0.65 mrem/year/pCi/L relates dose to concentration. From these values the alternatives of Table VIII-4 were generated.

For ^{228}Ra, as shown in Table VIII-2, the lifetime risk is 6.6 cases per pCi/L per million people exposed. The effective dose equivalent value of 0.38 mrem/year/pCi/L realtes dose to concentration. From these values the alternatives of Table VIII-4 were generated.

Allowing an uncertainty of a factor of four to five and rounding off all figures to one significant figure, the drinking water concentrations of ^{226}Ra and ^{228}Ra for different risk rates and EDEs are shown in Table VIII-3. The main source of uncertainty is from lack of precise knowledge of the pharmacokinetic parameters such as uptake. Other contributions include imprecision of the quality factor (Q) and the difference between

Table VIII-1 Calculations of the Effective Dose Equivalent Rate per pCi/L for Ingestion of ^{226}Ra

Organ	Lifetime risk/pCi/L/ million persons exposed[a]	70-Year dose equivalent rate/ pCi/L (mrem/year)[a,b]	Weighting factor[c]	Effective dose equivalent rate/ pCi/L (mrem/year)
Red bone marrow	3.74	1.68	0.16	0.269
Endosteal bone	0.88	19.86	0.009	0.180
Thyroid	0.42	0.24	0.099	0.024
Breast	0.68	0.24	0.13	0.031
Lungs	0.92	0.24	0.21	0.050
Stomach	0.44	0.24	0.084	0.020
Intestine	0.32	0.36	0.039	0.014
Liver	0.44	0.24	0.085	0.020
Pancreas	0.30	0.24	0.058	0.014
Urinary tract	0.12	0.24	0.025	0.006
Other	0.60	0.24	0.11	0.026
Totals	8.8			0.65

[a] Risk and dose equivalent calculations were done at the Oak Ridge National Laboratory, using the modified ICRP 30 model under an EPA contract.

[b] Assuming ingestion of 2 L/day.

[c] Derived by the USEPA (1984) from NAS (1980).

Table VIII-2 Calculations of the Effective Dose Equivalent Rate per pCi/L for Ingestion of ^{228}Ra

Organ	Lifetime risk/pCi/L million persons exposed[a]	70-Year dose equivalent rate/ pCi/l (mrem/year)[a,b]	Weighting factor[c]	Effective dose equivalent rate/ pCi/L (mrem/year)
Red bone marrow	1.96	0.71	0.16	0.114
Endosteal bone	0.39	7.02	0.009	0.063
Thyroid	0.42	0.24	0.099	0.024
Breast	0.67	0.24	0.13	0.031
Lungs	0.92	0.24	0.21	0.050
Stomach	0.43	0.24	0.084	0.020
Intestine	0.23	0.27	0.039	0.011
Liver	0.53	0.29	0.085	0.025
Pancreas	0.30	0.24	0.058	0.014
Urinary tract	0.13	0.25	0.025	0.006
Other	0.59	0.24	0.11	0.026
Totals	6.6			0.38

[a] Risk and dose equivalent calculations were done at the Oak Ridge National Laboratory, using the modified ICRP 30 model under an EPA contract.

[b] Assuming ingestion of 2 L/day.

[c] Derived by the USEPA (1984) from NAS (1980).

Table VIII-3 Lifetime Risk Rates and Annual Effective Dose Equivalent Rates as They Relate to Concentration of Radium in Drinking Water[a]

Lifetime individual risk rate	Annual effective dose equivalent rate (mrem/year)	Drinking water concentrations[b] (pCi/L)	
		^{226}Ra	^{228}Ra
10^{-3}	100	100	200
10^{-4}	10	10	20
10^{-5}	1	1	2
10^{-6}	0.1	0.1	0.2

[a] Assuming ingestion rate of 2 L/day.

[b] Rounded off to one significant figure and involving an uncertainty of a factor of four to five.

the estimates of the relative risk and the absolute risk models to relate risk to dose equivalent.

Table VIII-4 shows a suggested protective active guidance level for short-term exposures to radium by ingestion (calculated using the modified ICRP 30 dosimetric model). Here the dose equivalent refers to the total received for a lifetime.

E. Existing Standards

Table VIII-5 lists existing standards for ^{226}Ra from other countries. Some of the standards, targets or guidelines are lower than the 5 pCi/l MCL established as the interim regulations for U.S. drinking water.

F. Special Considerations

High-risk Subpopulations. There is no clear evidence of populations that are especially sensitive to radiotoxicity due to ingested radium.

Relative Radiotoxicity. As described in an earlier section, the relative radiotoxicity of different organs is accounted for in the dosimetric model. Thus, the estimated health effects to all organs can be summed using the weighting factors shown in Tables VIII-1 and VIII-2.

Table VIII-4 Concentration That Produces a Whole Body Dose of 500 mrem (pCi/L)

	1 day	10 days	1 year
^{226}Ra	5×10^5	5×10^4	1×10^3
^{228}Ra	9×10^5	9×10^4	2×10^3

Table VIII-5 Standards or Guidelines for ^{226}Ra in Drinking Water in the World

Country	Standard or guideline for ^{226}Ra
Australia	0.4 Bq/L (11 pCi/L)
Belgium	10 pCi/L (0.37 Bq/L)
Canada	27 pCi/L (1 Bq/L) (maximum acceptable, the target is 2.7 pCi/L)
Chile	3 x 10^{-12} microcuries/L (0.1 Bq/L or 2.7 pCi/L)
Guyana	2.7 pCi/L (0.1 Bq/L)
Hungary	3 x 10^{-9} micorcuries/mL (0.1 Bq/L or 2.7 pCi/L)
Iran	2.7 pCi/L (0.1 Bq/L)
Italy	sol. 0.1 x 10^{-7} microCi/mL (10 pCi/L) insol. 0.3 x 10^{-4} uCi/mL
Kuwait	0.27 pCi/L (0.01 Bq/L)
Republic of South Africa	1 microgram/L (recommended)
Romania	2.7 pCi/L (0.1 Bq/L)
Spain	4 x 10^{-8} microcurie/cc (40 pCi/L or 1.5 Bq/L)
USSR	1.2 x 10^{-10} microcuries/L (4.4 Bq/L or 120 pCi/L)

Adapted from Survey of the Uranium Task Force, Department of Health, Province of Nova Scotia, Canada (1984).

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APPENDIX

EPA POLICY STATEMENT ON RELATIONSHIP BETWEEN RADIATION DOSE AND EFFECT, MARCH 3, 1975[a]

The actions taken by the Environmental Protection Agency (EPA) to protect public health and the environment require that the impacts of contaminants in the environment or released into the environment be prudently examined. When these contaminants are radioactive materials and ionizing radiation, the most important impacts are those which ultimately affect human health. Therefore, the EPA believes that the public interest is best served by the Agency providing its best scientific estimates of such impacts in terms of potential ill health.

To provide such estimates, it is necessary that judgments be made which relate the presence of ionizing radiation or radioactive materials in the environment, i.e., potential exposure, to the intake of radioactive materials in the body, to the absorption of energy from the ionizing radiation of different qualities and, finally, to the potential effects on human health. In many situations, the levels of ionizing radiation or radioactive materials in the environment may be measured directly, but the determination of the resulting radiation doses to humans and their susceptible tissues is generally derived from pathway and metabolic models and calculations of energy absorbed.

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[a] From USEPA (1976).

It is also necessary to formulate the relationships between radiation dose and effects; these relationships are derived primarily from human epidemiological studies, but also reflect extensive research utilizing animals and other biological systems.

Although much is known about radiation dose-effect relationships at high dose levels, a great deal of uncertainty exists when high-level dose-effect relationships are extrapolated to lower dose levels, especially those involving lower dose rates. These uncertainties in the relationships between dose received and effect produced are recognized to relate to differences in quality and type of radiation, total dose, dose distribution, dose rate and radiosensitivity, including repair mechanisms, sex and variations in age, organ and state of health. These factors involve complex mechanisms of interaction among biological, chemical and physical systems, the study of which is part of the continuing endeavor to acquire new scientific knowledge.

Because of the many uncertainties, it is necessary to rely upon the considered judgments of experts on the biological effects of ionizing radiation. These findings are well documented in publications by the United Nations Scientific

Committee on the Effects of Atomic Radiation (UNSCEAR), the National Academy of Sciences (NAS), the International Commission on Radiological Protection (ICRP) and the National Council on Radiation Protection and Measurements (NCRP) and have been used by the EPA in formulating a policy on the relationship between radiation dose and effect.

It is the present policy of the EPA to assume a linear, non-threshold relationship between the magnitude of the radiation dose received at environmental levels of exposure and the ill health produced as a means of estimating the potential health impact of the actions it takes in developing radiation protection as expressed in criteria, guides or standards. This policy is adopted in conformity with the generally accepted assumptions that there is some potential ill health attributable to any exposure to ionizing radiation and that the magnitude of this potential ill health is directly proportional to the magnitude of the dose received.

In adopting this general policy, the Agency recognizes: (1) the inherent uncertainties that exist in estimating health impacts at the low levels of exposure and exposure rates which are expected to be present in the environment due to human activities and (2) that at these levels, the actual health impact will not be distinguishable from natural occurrences of ill health, either statistically or in the forms of ill health present. Also, at these very low levels, meaningful epidemiological studies to prove or disprove this relationship are difficult, if not practically impossible, to conduct. However, whenever new information is forthcoming, this policy will be reviewed and updated as necessary.

It is to be emphasized that this policy has been established to estimate the potential human health impact of Agency actions regarding radiation protection, and that such estimates do not necessarily constitute identifiable health consequences. Further, the implementation of this policy presupposes that, for the same dose, the potential radiation effects in other constituents of the biosphere will be no greater than those for humans. It is generally accepted that such constituents are no more radiosensitive than are humans. The Agency believes the policy to be a prudent one.

In estimating potential health effects, it is important to recognize that the exposures which will usually be experienced by the public will be annual doses that are small fractions of the natural background radiation, or at most, a few times this level. Within the U.S., the natural background radiation dose equivalent varies geographically between 40 to 300 mrem/year. Over such a relatively small dose range, any deviations from dose-effect linearity would not be expected to significantly affect actions taken by the Agency, unless a dose-effect threshold exists.

Utilization of a linear, non-threshold relationship is useful as a generally applicable policy for the assessment of radiation effects. However, in specific situations, it is also EPA's policy to utilize the best available detailed scientific knowledge in estimating health impact when such information is available for specific types of radiation, conditions of exposure and recipients of the exposure. In such situations, estimates may or may not be based on the assumptions of linearity and a non-threshold dose. In any case, the assumptions will be stated explicitly in any EPA radiation protection actions.

The linear hypothesis by itself precludes the development of acceptable levels of risk based solely on health considerations. Therefore, in establishing radiation protection positions, the Agency will weigh not only the health impact, but also the social, economic and other considerations associated with the activities addressed.

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